A Study of

CLINICAL STUDY ON ETIOLOGY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE DUE TO EXTRAHEPATIC BILIARY OBSTRUCTION

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CERTIFICATE

This is to certify that the dissertation entitled **" CLINICAL STUDY ON ETIOLOGY AND MANAGEMENT OBSTRUCTIVE JAUNDICE DUE TO EXTRA HEPATIC BILIARY OBSTRUCTION"** submitted by **Dr. A.SARAVANA KUMAR** to the Tamil Nadu Dr. M.G.R. Medical UniversityChennai in partial fulfillment of the requirement for the award of M.SDegree Branch – I (General Surgery) is a bonafide research work and was carried out by him under direct supervision & guidance.

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DECLARATION

I, **Dr. SARAVANA KUMAR A,** hereby declare that I carried out this work on,**" CLINICAL STUDY ON ETIOLOGY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE DUE TO EXTRAHEPATIC BILIARY OBSTRUCTION "**at the Department of general Surgery, Govt. Stanley medical college andHospital, chennai during the periodof aNOVEMBER2013 to OCTOBER 2015.under the guidance and supervision of my unit chief,**Prof.Dr.K.KUBERAN,Bsc.,MS., Professor of Surgery**I also declare that this bonafide work or a partof this work was not submitted by me or any others for any award, degree ordiploma to any other University, Board either in India or abroad.This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai inpartial fulfillment of the rules and regulations for the M.S degree examination inGeneral Surgery.

Place : CHENNAI **Dr.A.SARAVANAKUMAR**
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1.INTRODUCTION

Jaundice or icterus a generic term used for yellowish discoloration of the skin, mucous membrane or sclera caused by a heterogeneous group of disorders. It is useful to divide the causes of obstructive jaundice into two categories, cholestasis from parenchymal liver disease and mechanical obstruction from a block of the intrahepatic or extrahepatic biliary tract.

Surgical jaundice or Obstructive jaundice occurs due to the intra or extra hepatic obstruction to the biliary flow.

It can present as a problem in diagnosisand management because there is a group of jaundiced patients in whom it is very difficult to distinguish between organic / Structural obstruction and a medical cause of jaundice particularly intrahepatic cholestasis.

Biliary obstruction produces local effects on the bile ducts that lead to derangements of hepatic function and ultimately to widespread systemic effects. •

.Francis Glisson (1640), Abrahmson Vater (1720) and Ruggero Oddi (1887) refined anatomy with description of sphincteric mechanics.

• Charcot (1877) discussed the symptoms associated with

the passage of CBD stones which were jaundice, pain abdomen and fever (Charcot triad).

• Telfer Reynold added hypotension and altered mental status to Charcot's triad (Reynolds's pented) related to sepsis and cholangitis.

• Langenbach performed first cholecystectomy in the year 1882.

• Robert Abbe (1889) was the first to performed choledochotomy.

• Lawson Trait performer Choledocholithotomy.

• Ludwig Courvoisier (1843-1918) states Courvoisier's law.

Courvoisier Law:

In obstruction of the common bile duct due to a stone, distension of the gallbladder seldom occurs; the organ usually is already shrivelled. In obstruction from other causes the distension is

common. If there is no disease of gall bladder and the obstruction is due to a cancer of ampulla, pancreas and bile duct, then the gall bladder well may well distended.

• William Stewart Halstead performed Choledochoduodenal anastomosis for ampullaryCarcinoma.

• Emil Theodor Kocher's introduced Kocher incision and Kochermaneuverer.

• Charles Mcburney- Tran's duodenal Choledochotomy.

• Hans Kehr – Invented T-tube

• John B murphy – Cholecystoenterostomy avoiding choledochotomy

• The first mention of carcinoma gall bladder was published in 1777 in Ratio Mendendi of maximillian stall.

• Fredrich discussed Carcinoma gall bladder and suggested the relationship between gall bladder stone and cancer.

• Graham Cole (1925)- Oral cholecystography.

• Mirrizzi (1931) – Intra operative cholangiography.

• Okuda (1973) – CHIBA needle for percutaneous Transhepatic Cholangiography.

• Wildegans of Germany (1953) introduced modern choledochoscope.

Patients with complete biliary obstruction have clinical jaundice, whereas patients with intermittent biliary obstruction may present with pain, pruritus, fevers and biochemical changes without developing clinical jaundice. Patients with chronic incomplete obstruction eventually can develop hepatic fibrosis and biliarycirrhosis.

Two third of casesof obstructive jaundice are caused by congenital and benign diseases like calculus diseases of biliary tract, Choledochol cyst, pancreas divisum, annular pancreas, primary sclerosing cholangitis and post-operative or post pancreatitis strictures.

Malignant diseases like carcinoma head of pancreas,
Periampullary carcinoma, and cholangiocarcinoma and gall bladder malignancies are responsible for the rest.

Surgery as the modality of treatment for jaundice is not fully acceptable to a large majority ofpopulation in our part of the country. May be because of high belief in ayurvedic medicine,which is accepted as the best remedy for jaundice, and probably due to lower incidence ofobstructive jaundice in our population in the past. Anyhow there is an increasing evidence ofobstructive jaundice especially malignant obstructive jaundice. Surgeons thus face anincreasing number of patients with obstructive jaundice reaching them in a fairly advancestage.

The fundamental problem met with in dealing with a patient with prolonged jaundice is theaccurate diagnosis of its cause whether obstructive or not and if obstructive what exactly itscause.
In managing malignant obstructive jaundice the problem of diagnosis becomes an acute onebecause jaundice caused by mechanical obstruction to common bile duct should be surgicallyremedied whereas in the absence of mechanical block of bile duct treatment becomes medical.

The accurate diagnosis of mechanical obstruction to CBD becomes difficult at times becausethe clinical features and biochemical investigation may be atypical. Intrahepatic cholestasisgives rise to clinical features and laboratory data similar to mechanical block of common bile duct. Many times hepatocellular damage and mechanical obstruction coexist making thediagnosis much more difficult. Treatment of malignant obstructive jaundice is challenging. Surgical treatment ranges from definitive surgical procedures to palliative procedures. Non operative management includesendoscopic stenting, and interventional radiological procedure like PTBD. All these tests thesurgeon because of relative inaccessibility of the extrahepatic biliary tree and pancreas

2.REVIEW OF LITERATURE – I

EMBRYOLOGY OF LIVER AND BILIARY TRACT

Liver develops from an endodermal bud that arises from the ventral part of the junction between foregut and midgut. This bud grows into the ventral mesogastrium and passes into the septum transversum. This bud enlarges and divides into larger pars hepatica, and a smaller pars cystica. The pars hepatica divides into right and left parts and forms each lobe of liver. Sinusoids are formed from the mesenchyme of the septum transversum.

Bile formation begins in third month of gestation. The bile is responsible for the black colour of the first stools (meconium).

Gall bladder and Biliary passages:

The Gall Bladder and cystic duct develops from the pars cystica which divides from pars hepatica. The bile duct develops from the proximal part of the hepatic bud. The bile duct opens into ventral aspect of the developing duodenum. As a result of differential growth of the duodenal wall, and as a result of the rotation of the duodenalloop, the bile duct opens on the dorso-medial aspect of the duodenum along with the ventral pancreatic bud.

ANATOMY OF BILIARY TREE

The anatomy of the bile duct follows that of the portal system and segmentation of the liver. A bile duct is part of the portal triad, which enters the liver through invagination of Glisson's capsule at the hilum. According to the vascular anatomy, the right and left hemiliver are drained by a right and a left hepatic duct, respectively. Segment 1 is drained by several ducts joining both the right and left ducts close to the biliary confluence at the hilum. This anatomical knowledge is essential for hilar cholangiocarcinoma surgery. These hepatic ductsunite to form the common hepatic duct, which receives the cystic duct to become the common bile duct. The common bile ducts in most of the cases before entering the duodenum receivesthe pancreatic duct and the two share a final channel for about 7 mm in length within the pancreas. Thus the biliary tree has

1. Hepatic component
2. Extrahepatic component
3.Panncreatic component

Defects in the form of blockade anywhere in the hepatobiliary pancreatic system thus results
in cholestatic jaundice.

GROSS ANATOMY

LIVER

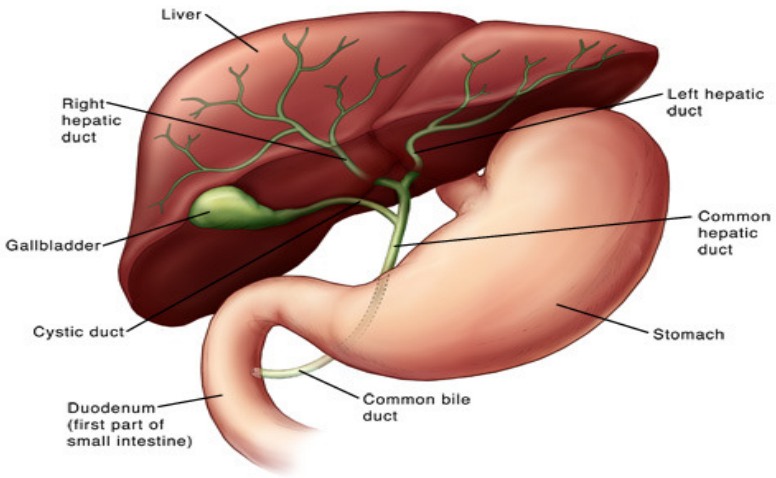
Liver is the largest gland in the body. It is almost completely covered with peritoneum, whichis reflected on to the adjacent structures forming the so called falciform ligament of the liver.

The region devoid of peritoneum is the bare area of the liver. The liver has got diaphragmatic and visceral surfaces. The diaphragmatic surface has anterior,posterior, superior and lateral surface or parts.

The bare area of liver comes in the posterior
surface of the liver. The porta hepatis and the gall bladder are related to the visceral or inferiorsurface.

Grossly the liver is divided into a larger right and a smaller left lobe by the attachment of thefalciform ligament on the superior and anterior surface of the liver and the groove for

ligamentum venosum on the posterior surface. Thus the anatomical right lobe includes thequadrate lobe on the inferior surface and caudate lobe on the posterior



figure;1

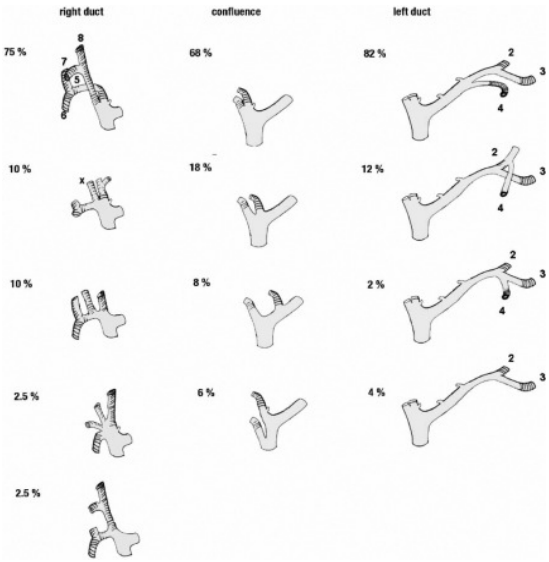
From the surgical point of view the more important one is the functional right and left lobesof the liver, the dividing line pass through the gall bladder bed anteriorly and inferiorly and through the groove for the inferior vena cava posteriorly. This functional division is based onthe territory of arterial supply, venous and biliary drainage. Thus the caudate and quadrate

lobes belong to the functional left lobe of the liver. The left hepatic duct drains segments 2, 3, and 4 of the left hemiliver. The “normal” confluence comprises a duct formed from ducts of segments 2 and 3 and one or more ducts from segment 4. The segment 3 duct follows the left horn of the Rex recessus and joins the segment 2 duct above the segment 2 portal branch (at the level of the curve of the hilar part in the posterior-anterior portion of the portal branch). This duct is 2.5 cm long, from 2 to 5 cm, depending on the size of the posterior margin of the quadrate lobe. Being extrahepatic in this portion, it runs transversely in the hilum, from left to right. Running first above and behind the left portal branch, it crosses the superior edge and joins the right hepatic duct to form the biliary confluence. For the left hepatic duct [1], this normal anatomy is reported in 82%. In 4% of patients, a right sectoral duct can join the left hepatic duct (3% posterior and 1% anterior).

The right hepatic duct drains all segments of the right hemiliver (segments 5, 6, 7, and 8). The ducts of segments 6 and 7 form the posterior right hepatic duct, and those of segments 5 and 8 form the anterior right hepatic duct. The anterior hepatic duct lies vertical,

located to the left of the anterior branch of the portal vein. The direction of the posterior duct is more horizontal, running superior (being epiportal in the Hjortsjö crook)to the anterior portal branch and joins the anterior duct. In approximately 20% of instances, the right duct runs inferiorly (being hypoportal) to the anterior portal branch. A complete anterior duct was present in 35% and a complete posterior duct in 61%. The “normal” confluence [1] of these two ducts forms the right hepatic duct, above the right portal branch, in an extra-hepatic position. The right hepatic duct can be absent, the anterior and posterior ducts joining directly to the left hepatic duct, forming a triple confluence (12%). The right hepatic duct may join the main hepatic duct below the normal confluence in 25% of cases (9% the anterior and 16% the posterior). This anatomical variation is known as “convergence étagée” or selved confluence. The normal right duct is short and vertical and 1 cm in length

Biliary confluence



figure;2

The main biliary confluence is formed outside the liver parenchyma, before becoming distal to the common hepatic duct. It runs along and anterior to the origin of the right branch of the portal vein The duct is displaced superiorly and medially to the left of the main portal vein. This classic junction occurs in 61% of instances. During a right hepatectomy, the anatomical situation of the main biliary confluence explains the risk of ligating the confluence or the left duct. The Bismuth–Corlette classification [5] is valid only for a “normal” confluence. In the event of biliary abnormality, it is necessary to take

into account not only the type of confluence, but also its height in relation to the portal vein.

At the level of the hilum, Glisson's capsule is both thicker and denser, forming the connective tissue of the hilar plate. The biliary ducts are enclosed within this tissue. Adhesions between this capsule and arterial and portal branches are less important. It is therefore easy to dissect the portal branches at the hilum, but more difficult for the arterial branches and almost impossible to separate the bile duct of the hilar plate .In the case of hilar cholangiocarcinoma, the proximity of the portal triad explains the frequent tumor invasion of portal branches. A lobar atrophy may result from the vascular invasion and/or from a biliary obstruction. Owing to the absence of vascular interposition at the anterior part of the hilar plate, it is also possible to separate the hilar plate and hepatic parenchyma of segment.

Gall Bladder:

The gallbladder is 7-10 cm long and has a capacity of 30-50 ml.
It is located on the visceral surface of the liver in a shallow fossa at the plane dividing the right lobe from the medial segment of the left lobe (the GB-IVC line). In other words, the gallbladder fossa is found at the junction of the quadrate lobe (segment IV) and the right lobe of the liver along the line of Rex. The gallbladder is separated from the liver by the connective tissue of Glisson's capsule. Anteriorly, the peritoneum of the gallbladder is continuous with that of the liver.

The gallbladder can be divided into fundus, body, infundibulum, neck, and cystic duct.

The fundus projects beyond the inferior margin of the liver, in the angle between the lateral border of the right rectus abdominis and the ninth costal cartilage. It is entirely surrounded by peritoneum
The body lies in the fossa for the gall bladder on the liver. The

upper narrow part of the body is continuous with the neck at the right end of the porta hepatis. The superior surface of the body is devoid of peritoneum, and is adherent to the liver. The inferior surface is covered with peritoneum, and is related to proximal part of transverse colon and first part of the duodenum.

The neck is the narrow upper end of the gall bladder. It first curves anterosuperiorly and then posteroinferiorly to become continuous with the cystic duct. Its junction with the cystic duct is marked by a constriction. The posteromedial wall of the neck is dilated to form a pouch called the Hartmann's pouch which is directed downwards and backwards.

Cystic Duct:

Cystic duct is about 3 to 4 cm in length. It ends by joining with the common hepatic duct at an acute angle to form the common bile duct. The mucous membrane of the cystic duct forms a series of 5 to 10 crescentic folds, arranged spirally to form the so-called "*spiral valve" of Heister*. This is not a true valve.

Common Bile Duct:

The common bile duct begins at the union of the cystic and common hepatic ducts and ends at the papilla of Vater in the second part of the duodenum. It varies in length from 5 cm to 15 cm, depending on the actual position of the ductal union. In 22%, the common hepatic and cystic ducts, on average, run parallel for 17 mm before the ducts actually unite. The average diameter is about 6 mm
The common bile duct can be divided into four portions or segments: supraduodenal, retroduodenal, pancreatic, and intramural.
The supraduodenal portion of the common bile duct lies between the layers of the hepatoduodenal ligament in front of the epiploic foramen of Winslow, to the right or left of the hepatic artery, and anterior to the portal vein. Its length is 2-5 cm.
The distal part of the supraduodenal portion is related to the posterior superior pancreaticoduodenal (PSPD) artery, which has a retroduodenal location and which crosses the duct first anteriorly and then posteriorly. This artery is not to be confused with the

supraduodenal artery, which also may pass anterior to the common bile duct. In the majority of cases the retroportal artery joins the PSPD artery, but it may join the right hepatic artery directly and send branches to the common duct en route. The PSPD artery is easily injured while exploring the common duct.

If the junction of the cystic and common hepatic ducts is low, the supraduodenal segment is short or even absent. Large lymph nodes may be fixed to the right side of the supraduodenal segment.

The retroduodenal portion of the common bile duct is between the superior margin of the first portion of the duodenum and the superior margin of the head of the pancreas. It is 1-3.5 cm long. The duct may be free or partially fixed to the duodenum11.

The pancreatic portion of the common bile duct extends from the upper margin of the head of the pancreas to the point of entrance into the duodenum. It passes downward to the right, posterior to the pancreas or within the pancreatic parenchyma.

The intramural portion of the common bile duct takes an oblique path averaging 1.5 cm through the duodenal wall. Here it

receives the main pancreatic duct inferiorly. The two ducts usually lie side-by-side with a common adventitia for several millimetres. The diameter of both ducts decreases within the duodenal wall. The septum between the ducts is reduced to a thin mucosal membrane before the ducts become confluent3 11.

The terminal part of the bile duct is surrounded just above its junction with the pancreatic duct by a ring of smooth muscle that forms the sphincter choledochus. This sphincter is always present. It keeps the lower end of the bile duct in closed status. As a result, bile formed in the liver keeps accumulating in the gall bladder and also undergoes considerable concentration. When food enters the duodenum, especially a fatty meal, the sphincter opens and the bile stored in the gall bladder is poured into the duodenum.

Another less developed sphincter, which is usually but not always present around the terminal part of the pancreatic duct, is called sphincter pancreaticus. A third sphincter surrounds thehepatopancreatic ampulla and is called the sphincter ampullae. The sphincter ampullae may extend proximally to enclose the lower parts of bile and pancreatic ducts.

The sphincters named above are often referred to collectively as *the sphincter of Oddi*.

PANCREAS

Pancreas is a retroperitoneal elongated gland lying between the C loop of duodenum and

splenic hilum. It has a head, body and tail with a small constricted part between head andbody called the neck and another small downward projection from the head called the

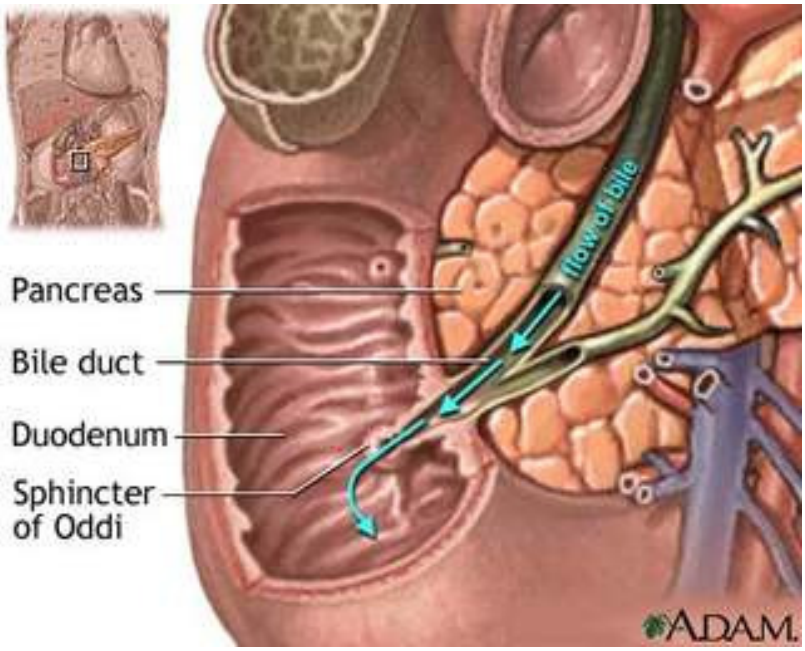


Figure ;3shows :Flow of bile through ampulla of vater

Uncinate Process. The main ductal system of the pancreas, the Duct of Wirsung, starts fromthe tail lying near the posterior than the anterior surface with small ducts of the lobes draining
to it at right angles forming a herring bone pattern. It traverses the body and on reaching theneck it binds down posteriorly to join with the common bile duct to form a common dilatedhepatico pancreatic Ampulla of Vater surrounded by the Sphincter of Oddi which prevents thereflux of bile into the pancreas and vice versa.

Blood supply:

The cystic artery arises from the right hepatic artery as it crosses the calot's triangle to the right of the common hepatic duct. The lymph node of Lund usually lies just superficial to the position of the cystic artery in the cystic triangle, and can be a good guide to finding and ligating it. Reaching the gallbladder behind the common hepatic duct, the cystic artery usually branches into an anterior superficial branch and a posterior deep branch. These branches anastomose and send arterial twigs to the adjacent liver. The cystic artery may arise from the left hepatic artery or the gastroduodenal artery
The extrahepatic bile ducts in most individuals are supplied

from the cystic artery above and from the posterior superior pancreaticoduodenal artery below.

The epicholedochal arterial plexus of the CBD is derived from the retroduodenal or pancreaticoduodenal arteries. The collateral circulation is enhanced by two intramural plexuses. These may be compressed between the oedematous mucosa and the external tough fibrous coat in pathologic conditions such as cholangitis or common bile duct obstruction secondary to choledocholithiasis.

Veins:

The superior surface of the gallbladder is drained by multiple small veins passing through the gallbladder bed that breaks up into capillaries within the liver. They do not form a single "cystic vein." Veins from the hepatic surface drain directly into the liver. Veins on the inferior surface open directly or follow the hepatic ducts into the liver. From the peritoneal surface, one vein usually drains the fundus and body and other veins drain the neck and upper portions of the cystic duct as well as the hepatic ducts. These small veins enter the liver together with ascending veins from the common bile duct. These veins rarely open into extra hepatic portal veins.

Nerve Supply:

• Coeliac plexus

• Seven to nine thoracic sympathetic fibres

Pain from the gall bladder may travel along the vagus, the sympathetic nerves, or along the phrenic nerves. It may be referred to different parts through these nerves as follows.

1. Through the vagus to the stomach

2. Through the sympathetic nerves to the lower pole of the scapula

Calot Triangle:

It is bounded, right side by the upper part of the gallbladder and cystic duct, left side by the common hepatic duct and superiorly by the inferior surface of the right lobe of the liver.

Histology:

The bile ducts are composed of an external fibrous layer of connective tissue, a few thin smooth muscle layers (longitudinal, oblique, and circular), and an internal layer of mucosa of columnar epithelium. The gallbladder wall is formed, from external to internal, by the following layers:

Serosa

Adventitia

Fibro muscular layers

Mucosa

Serosa is the typical visceral peritoneum formed by mesothelium on the surface with loose connective tissue directly beneath. Adventitia is a layer of dense connective tissue that is found external to the muscularis externa where the gallbladder is attached to the surface of the liver. The adventitia contains large blood vessels, autonomic fibres for innervation of muscularis externa and blood vessels, a rich lymphatic network, and a plethora of elastic fibre's and adipose tissue.

Fibro muscular layers comprise many elastic and collagen

fibres among bundles of smooth muscle cells. No muscularis mucosa or submucosa is found in the gallbladder. Mucosa is distinguished by having very tall, slender columnar epithelial cells. While no glands are found in the mucosa, this layer is thrown into elaborate folds which on first inspection give the impression of glands. These folds form deep diverticula of the mucosa and have been identified as "Rokitansky-Aschoff sinuses"; in some cases, these extend through the muscularis externa. Bacteria have been known to accumulate in these folds, and chronic inflammation may develop.

Physiology:

Bile produced by hepatocytes, drains into the hepatic canaliculi.
It travels from the terminal bile ducts to the right and left hepatic ducts. Then it moves to the common hepatic duct. The majority of the bile goes from the common hepatic duct through the cystic duct to the gallbladder, drains to the common bile duct, and then to the duodenum. The remainder of the bile goes to the common bile duct, then to the duodenum, bypassing the gallbladder.
Bile production is such that 250 ml to 1,500 ml of bile enters

the duodenum each day. The gallbladder has a capacity ranging from 15 to 60 ml (average approximately 35 ml). The gallbladder concentrates bile by absorbing sodium, chloride, and bicarbonate ions and water such that bile salts can be concentrated 5 to 250 times. Potassium ions are concentrated as the water is absorbed; further concentration results from simple diffusion. Bile contains significant amounts of carbonate and calcium ions. The epithelium secretes hydrogen ions, and the carbonate ions are converted to bicarbonate. Calcium and bicarbonate ions are absorbed by the epithelial cells and, thus, calcium carbonate precipitation in the gallbladder is avoided9. The hormone cholecystokinin causes contraction of the gallbladder muscle, forcing bile out. Stimulation from the vagus nerve also causes the gallbladder to contract. The sphincteric apparatus of Oddi becomes inhibited in the presence of cholecystokinin and relaxes as a reaction to gallbladder contraction. All of these actions force bile into the CBDandintothe second part of duodenum.

Physiology of the Gallbladder and Bile Ducts

The anatomy of the biliary tree is a little complicated, but it is important to understand. The liver's cells (hepatocytes) excrete bile into canaliculi, which are intercellular spaces between the liver cells.

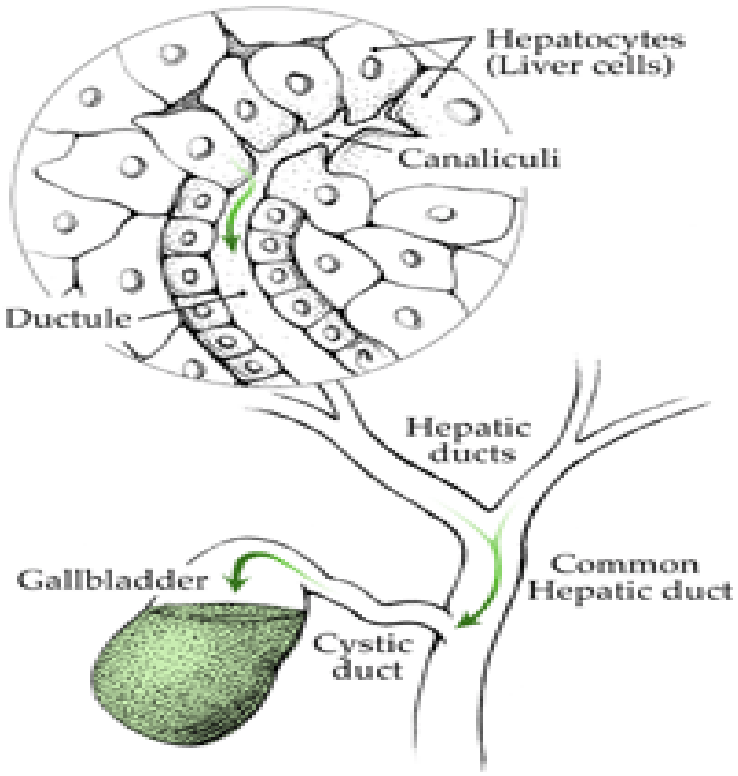


Figure 4; shows phsiological flow pattern of bile

These drain into the right and left hepatic ducts, after which bile travels via the common hepatic and cystic ducts to the gallbladder. The gallbladder, which has a capacity of 50 milliliters (about 5 tablespoons), concentrates the bile 10 fold by removing water and stores it until a person eats. At this time, bile is discharged from the gallbladder via the cystic duct into the common bile duct and then into the duodenum (the first part of the small intestine), where it begins to dissolve the fat in ingested food.

The liver excretes approximately 500 to 1000 milliliters (50 to 100 tablespoons) of bile each day. Most (95%) of the bile that has entered the intestines is resorbed in the last part of the small intestine (known as the terminal ileum), and returned to the liver for reuse.

The many functions of bile are best understood by knowing the composition of bile:

Bile Salts (cholates, chenodeoxycholate, deoxycholate): these are produced by the liver's breakdown of cholesterol. They function in bile as detergents that dissolve dietary fat and allow it to be absorbed. Hence, disruption of bile excretion disrupts the normal absorption of fat, a process called malabsorption. Patients develop diarrhea because

the fat is not absorbed (steatorrhea) , and develop deficiencies of the fat-soluble vitamins (A, D, E, and K).

Cholesterol and phospholipids-while only 4% of bile is cholesterol, the secretion of cholesterol and its metabolites (bile salts) into bile is the body's major route of elimination of cholesterol. Phospholipids, which are components of cell membranes, enhance the cholesterol solubilizing properties of bile salts. Inefficient excretion of cholesterol can cause an increased serum cholesterol. This predisposes to vascular disease (heart attacks, strokes, etc.)

Bilirubin-while this comprises only 0.3% of bile, it is responsible for bile's yellow color. Bilirubin is a product of the body's metabolism of hemoglobin, the carrier of oxygen in red blood cells. Disruption of the excretion of this component of bile leads to a yellow discoloration of the eyes and skin (jaundice).

Protein and miscellaneous components

Bile production and recirculation is the main **excretory function** of the liver. Tumors that obstruct the flow of bile from the liver can also impair other liver functions. Therefore, it is necessary to understand

these other functions to understand the symptoms that these tumors can cause. These include:

Metabolic functions, such as the maintenance of glucose (blood sugar) levels

Synthetic functions, such as the synthesis of serum proteins such as albumin, blood clotting (coagulation) factors, and complement (a mediator of inflammatory responses)

Storage functions, such as the storage of sugar (glycogen), fat (triglycerides), iron, copper, and fat soluble vitamins (A, D, E, and K)

Catabolic functions, such as the detoxification of drugs

CHAPTER III
3.REVIEW OF LITERATURE – II

JAUNDICE

The term 'Jaundice' is derived from the French word meaning 'Yellow' and refers to the presence of an excess of bile pigments in the tissues and the serum. It is a presenting sign of anumber of hepatic and non-hepatic diseases. The differential diagnosis and management aredependent upon a appreciation of normal and abnormal variants of bile pigment metabolism.

PATHOPHYSIOLOGICAL CLASSIFICATION OF JAUNDICE

I. PREDOMINANTLY UNCONJUGATED HYPERBILIRUBINEMIA

A. Excess production of bilirubin

1. Hemolytic anaemia
2. Resorption of blood from large internal hemorrhages
3. Ineffective erythropoiesis

B. Reduced hepatic uptake

1. Drug induced
2. Prolonged fasting
3. Sepsis

C. Impaired bilirubin conjugation

1. Gilbert's Syndrome
2. Criggler-Najjar Syndrome I & II
3. Physiological jaundice of new born

4. Diffuse hepatocellular disease (hepatitis, cirrhosis)

II. PREDOMINANTLY CONJUGATED HYPERBILIRUBINEMIA (CHOLESTATIC JAUNDICE)

increase in serum bilirubin is in the unocnjugated indirect reacting bilirubin, no bilirubinappears in the urine but there is an increase in the fecal and urinary urobilinogen. An excessof bilirubin production also occurs in shunt hyperbilirubinemia in which indirect bilirubinaccumulates in the absence of any reduction in red cell life span.Constitutional defects of liver function may also cause hyperbilirubinemia withoutimpairment of bile flow. In Gilbert's disease there is defect in the bilirubin transport into theliver cell, while in Criggler-Najjar syndrome the defect is an inability of liver to conjugate the bilirubin with glucuronic acid. In these states, the elevation of bile pigments is in the indirectreacting fraction. All other hepatic function tests are normal, and no histological abnormality is noted with all of the above.

A. Decreased intrahepatic excretion of bilirubin

1. Dubin Johnson Syndrome

2. Rotor's Syndrome

3. Drug induced

4. Hepatocellular disease (viral hepatitis)

5. Primary biliary cirrhosis

6. Sclerosing Cholangitis

B. Extrahepatic biliary obstruction

1. CBD stones

2. Carcinoma of the head of pancreas, extrahepatic hile ducts and ampulla of Vater

3. Extrahepatic biliary atresia

NORMAL BILE PIGMENT METABOLISM

The bile pigment – bilirubin is a tetra pyrrole, which is formed to the greatest extent fromhemoglobin and to a lesser extent from myoglobin breakdown and hepatic synthesis itself.

When the red blood cell is destroyed by the reticuloendothelial system the iron and globin are

removed and the heme ring is opened and transformed into biliverdin, which is green. Thelater is reduced to become bilirubin, which is yellow. The bilirubin combines with albumin toform a relatively stable protein-pigment complex and is transported as such to the hepaticparenchymal cell. This complex which is referred to as indirect reacting bilirubin, since itgives the Vanderbergh diazo reaction only after treatment with alcohol and other substance
that split the protein, is poorly soluble in water and is not excreted in the urine. In the hepatic parenchymal cell the albumin is removed and the bilirubin is conjugated withglucuronic acid to form diglucuronide, which is water soluble and is excreted into the biliarycanaliculi. This substance gives an immediate diazo reaction and hence termed as directreacting.

This is passed into urine. Normally there is less than 1.2 mg of direct reacting serumbilirubin and less than 0.3 mg of indirect reacting bilirubin per 100 ml of serum.

The conjugated bilirubin which is excreted via the bile into the intestine is acted upon bybacteria and undergoes a series of reduction leading to the formation of two groups ofcompounds namely the colourless urobilinogen and coloured Stercobilin. The normal dailyfecal excretion ranges between 40 and 300 mg with an average of 100-200 mg, and innewborn infants because of the absence of bacterial flora urobilinogen may be absent. Areduction in the enteric bacteria is also responsible for the reduced pigment excretion that

accompanies the use of intestinal antibiotics. Some of the urobilinogen is reabsorbed by wayof portal venous system and returns to the liver, where it is either removed or to a small extentexcreted in urine.

ABNORMAL BILE PIGMENT METABOLISM

No classification is totally satisfactory. The classification most widely used distinguishesbetween hemolytic, obstructive and hepatocellular jaundice. However, it is most reasonable to

categorize as

1. Those disease states in which the bile flow is unimpeded.

2. Those types that are associated with an impairment of the bile flow.

NORMAL BILE EXCRETION

The overproduction of bile pigment from excessive hemolysis creates a situation in which

normal liver is confronted with more pigment than it is able to remove. This occurs inphysiological jaundice of infancy and all pathological hemolytic states. However the reservecapacity of the liver is great and even when the bilirubin production is increased six times

there is only a 2-3 mg rise in the serum bilirubin level per dL of serum. In this situation thementioned diseases the bilirubin pigment is attached to

albumin and cannot be excreted by the kidney, thus prompting the term acholuric jaundice.

IMPAIRED BILE EXCRETION

All other diseases are associated with an accumulation of conjugated bilirubin in the bloodand impaired excretion. The bilirubin pigment which is water soluble, is readily excreted into the urine, which becomes brown. The obstruction may be intrahepatic or extrahepatic.

INTRAHEPATIC OBSTRUCTIVE JAUNDICE

In the Dubin-Johnson Syndrome, which is associated with the appearance of iron free pigmentin the hepatic cells and normal liver function, the hepatic excretion of conjugated bilirubin isimpaired. Intrahepatic cholestasis has also been related to a variety of drughepatocellular disease. Methyltestosterone and norethiandrolene damage the microvilli of the
bile canaliculi and may cause jaundice. The phenothiazine drugs such as chlorpromazine mayevoke a hypersensitivity reaction in a small percentage of patients and result in cholangitichepatitis and intrahepatic cholestasis. A lesion along the excretory pathway within the liver is
believed to cause obstructive jaundice associated with primary biliary cirrhosis.

EXTRAHEPATIC CHOLESTASIS

This is caused by anatomical obstruction to flow of bile from liver to the intestine. Theobstacle may be situated anywhere from the junction of right and left hepatic ducts to thetermination of common bile duct in the duodenum. Atresia, stricture, choledocholithiasis,tumours of bile duct and pancreas, choledochal cysts and parasites have been implicated.

Obstruction of extrahepatic duct results in an increase in serum bilirubin particularly thedirect reacting type, the appearance of bile in the urine and passage of clay coloured stools.

When total bilirubin level is above 3 mg/dL, the increase in both the direct and indirectreacting fraction parallel one another. With complete and persistant obstruction the serumbilirubin may plateau. In the fluctuating obstruction levels will change.

EFFECTS OF BILIARY TRACT OBSTRUCTION

PHYSICAL EFFECTS

The normal secretary pressure of bile is 120-250 mm of water. Following total bile ductobstruction, bile secretion will continue until CBD pressure rises to 170-220 mm of waterafter which secretion decreases. Cholesterol and phopholipid secretion is more readilyreduced by high pressure than bile salt secretion making bile less lithogenic.

Complete obstruction of main extrahepatic bile duct of major segmental duct will normallylead to proximal dilatation. The lack of intrahepatic dilatation may be due to seconday hepaticfibrosis of co-existing alcoholic cirrhosis.

PAIN

Painless progressive jaundice is the classical hallmark of malignant biliary tract obstruction.But it is not uncommon to elicit a history of abdominal pain in these patients,the cause ofpain being distention of gall bladder and bile duct or associated stretching of liver capsule inrapidly progressive obstruction.

PATHOLOGICAL CHANGES IN BILE DUCTS AND CANALICULI

In biliary obstruction the canaliculi become dilated and microvilli distorted and swollen. Bilepigment thrombi may be seen in canaliculi and adjacent hepatocytes. In prolonged cholestasis,the canaliculi increase in length and tortuosity. Resorption of bile constituents from ductules

leads to marked inflammatory reaction in the portal tracts with polymorphonuclear leucocyteinfiltrate. The hepatocyte of periportal zone shows disruption and eventually leading on topiecemeal necrosis. Experimental evidence shows that if obstruction is relieved within
weeks morphological changes are reversible.

CHOLANGITIS

Although the neutrophil associated with cholangitis is a chemical reaction associated withbiliary obstruction and does not imply bacterial inflammation, in presence of biliary stasis,secondary bacterial colonization may produce the additional element of infective cholangitisalthough classically referred to as ascending cholangitis the actual mechanism for entry ofbacteria into the unoperated biliary tract may not always be clear. Studies by McPherson et al (1982) showed that organisms are found in bile in approximately 1/3$_{rd}$ of patients withmalignant biliary enteric anastamosis this rate may be higher. In another study Jackaman et al(1980) found that highest rate of biliary colonization were found in patients withcholedocholithiasis and benign bile duct strictures where as may as 80% of patients hadpositive cultures.

ATROPHY

The characteristic effect of unilateral hepatic duct obstruction is atrophy of obstructed liverparenchyma with compensatory hyperplasia of unaffected segments of liver. A grosslyhypertrophied left lobe palpable in association withunilateral obstruction may not producesufficient hyperbilirubinemia in

presence of normal contralateral lobe.The practical importance of lobar atrophy in a surgical context lies in the fact that an atrophicliver lobe may be inadequate to support life following the resection of normal or hyperplastic
liver tissue and biliary drainage of such an obstructed lobe may also fail to produce resolutionof jaundice.

BIOCHEMICAL EFFECTS

BILIRUBIN

Conjugated hyperbilirubinemia is the classical biochemical feature of obstructive jaundice.But prolonged partial obstruction with functional effects on hepatocytes may produce a mixedbiochemical picture with elevated circulating unconjugated bilirubin.

ALKALINE PHOSPHATASE

Elevation of this enzyme is the most widely used and probably the most sensitive indicator. Itmay be the only biochemical indicator of incomplete of segmental obstruction. Acuteobstruction of bile duct causes regurgitation of enzyme from biliary compartment andincrease in hepatic synthesis.

PROTEIN SYNTHESIS

Liver occupies a central role in protein synthesis and quantitatively albumin is the mostimportant protein synthesized by liver. However due to its long half life (20 days) onlyminimal changes occur to hepatocyte damage. Nonetheless the

frequent association of biliaryobstruction with malignancy and malnutrition causes hypoalbuminemia.An active marker of hepatic protein synthesis, serum prealbumin is more valuable since it hashalf life of only 1.9 days. The most important aspect of protein synthesis relates to synthesis
of coagulation factors II, VII, IX & X and its failure is due to failure to absorb vit K due toabsence of bile salts from intestine.

LIPIDS

Cholesterol level may be elevated in biliary tract obstruction. A number of alterations in lowdensity lipoproteins have been observed which are of no major functional importance.

CARBOHYDRATE METABOLISM

Abnormal glucose tolerance may be seen in patients with impaired liver function. But themalignant disease causing obstruction might be primary cause.

BILE SALT CIRCULATION

The enterohepatic circulation of bile salts is completely interrupted by total biliaryobstruction. This may lead to gross elevation of serum bile acid levels. This has two importantmetabolic consequences. Firstly, due to the absence of bile salts in the intestine the smallbowel microflora gets altered. Secondly, following external biliary drainage, the secretion ofbile is under altered physiological drive.

ENDOTOXEMIA & RETICULOENDOTHELIAL FUNCTION

Endotoxin is a lipopolysaccharide derived from the cell walls of gram negative bacteriapresent in the gut. Normally only minute quantities of endotoxin enters the portal circulationand these traces are cleared by hepatic reticuloendothelial system. In obstructive jaundice theabsence of bile salts from intestine causes increased formation of endotoxin by alteredmicroflora and decreased clearance of absorbed endotoxin due to depressedreticuloendothelial cell function resulting in endotoxemia in more than 50% of patients. The bile salt absorption is also quiet fast in biliary obstruction probably due to increased vascularpermeability. The circulating endotoxin causes pathological effects like renalvasoconstriction, redistribution of intrarenal blood flow and activation of complement,leukocytes and platelets.

CHANGES AFTER RELIEF OF OBSTRUCTION

BILE SECRETION

Postoperative study of biliary secretion is done by the insertion of external percutaneoustranshepatic drain. There is frequently a prompt and major cholestasis and bile volumes mayexceed 4 liters per day. Failure to replace large volumes of fluid and electrolyte losses mayresult in dehydration and electrolyte depletion with a metabolic acidosis. The replacement of bile salts if desired may also be undertaken in the form of commercially available preparation.

During the first few days of biliary drainage the bile produced is of low bilirubin and bile saltconcentration. This may be partly due to a slow return of impaired liver to normal functionand also to loss of enterohepatic circulation of bile salts.

RECOVERY OF FUNCTION

In majority of case plasma bilirubin begins to fall promptly after insertion of a drainageIn majority of case plasma bilirubin begins to fall promptly after insertion of a drainagecatheter or an internal biliary bypass procedure and this is accompanied by clinical . However, return of hepatocyte function is not instantaneous.Assessment of liver function by serial antipyrene clearance measurement after relief ofobstruction has shown that it takes about 6 weeks for it to return to normal values.

STRUCTURAL CHANGES

The reversal of structural changes in liver and biliary tract following decompression ofobstruction is variable. Bile ducts which have beeen subjected to edema, inflammatoryinfiltration, cholangitis, and fibrotic changes are likely to retain some rigidity for considerabletime after decompression.

As regards reversal of intra hepatic fibrotic changes following drainage it is difficult to obtainclear evidence since this would rely upon serial liver biopsies in asymptomatic patients solong as fibrotic changes remain short of true secondary biliary cirrhosis, they are reversible byadequate drainage. Even the portal hypertension secondary to such fibrosis may be improved

with such drainage.

CLINICAL FEATURES

SYMPTOMS

Typically a patient with obstructive jaundice presents with dark urine, pale stools and pruritusof varying severity. Information regarding initial onset and whether clinical course isintermittent and associated with pain, fever and rigors must be short. Attack precipitated byfat intake can be relevant. An episode of cholangitis is recognized if jaundice is associatedwith pain, rigor and pyrexia. Jaundice without significant pain or pain radiating to back may indicate pancreatic pathology. However this is not certain and patients with gallstones anpresent with back pain whereas patients with extensive carcinoma of head of pancreas may present with typical history of biliary colic.

A fluctuating depth of jaundice is suggestive of intermittent obstruction as in periampullarycarcinoma or temporary alteration of stone in the ampulla of vater. It is very rare in pancreaticcancer and cholangiocarcinoma. Weight loss, anorexia and pallor suggest malignancy of shortduration. When these symptoms occur with painless jaundice, neoplasm of head of pancreas is likely. Pruritis may be present in all forms of jaundice and may either be progressive orfluctuate in intensity.

PHYSICAL EXAMINATION

GENERAL INSPECTION

The common stigmata of liver disease should be looked for – they are all indications of liverdysfunction. Jaundice is due to staining of tissues with bilirubin and possibly other pigmentssuch as biliverdin. It is initially noticed in sclera. As jaundice progresses the skin becomesprogressively more pigmented, spider naevi, which are vascular skin lesions supplied bycentral arteriole is occluded with a pinhead. Spider naevi usually occur in the region of superior vena cava – chest above the level of nipple, face, neck and arms. Palmar erythema isobvious and pronounced reddish flushing of palms. It particularly affects the thenar and hypothenar eminece and bases of fingers. Spontaneous bruising, echymosis and bleedingaround venipuncture sites are well recognized signs of liver disease occurring due toabnormality in coagulation mechanisms. Long standing pruritis causing scratch marks all over the body can also be noted.

EXAMINATION OF LIVER

Palpation of liver should be combined with percussion to determine the upper and lowerborders. The upper border of liver normally extends upto 5th intercostals space. Auscultationover the liver may give some evidence of underlying disease. An arterial bruit is evidence ofhepatocellular carcinoma and venous hum in portal hypertension.

SPLENIC ENLARGEMENT

Splenomegaly can be detected by palpation commencing in the right iliac fossa andprogressing towards the left hypochondrium. Splenic notch can sometimes be recognized onthe anterior border of grossly enlarged spleen.

ASCITIS

Clinical confirmation of ascitis is achieved by eliciting shifting dullness on percussion or fluidthrill on palpating the flanks. Ascitis could be due to hypoalbuminemia of liver dysfunction,portal hypertension or manifestation of advanced malignancy either of liver or pancreas.

GALL BLADDER SIGNS

The finding of a palpable gall bladder in the presence of features of obstructive jaundicesuggests malignant obstruction of the biliary tree (Courvoisier's Law). However failure topalpate gall bladder does not exclude the presence of malignant biliary obstruction. On theother hand it is possible to have a palpable gall bladder in the presence of gallstones whereone stone obstructs the common bile duct and another is impacted in the Hartmann's pouch or cystic duct resulting in an empyema or mucocele of the gall bladder. An intermittentlypalpable gall bladder is suggestive of periampullary carcinoma.

EVIDENCE OF PORTAL HYPERTENSION

Portal hypertension is usually associated with hepatosplenomegaly and ascitis. Large dilatedabdominal wall veins occur due to collateral circulation between the portal system andsystemic veins.

DIFFERENTIAL DIAGNOSIS IN CHOLESTASIS

I. EXTRAHEPATIC CAUSES

1. STONES

a) gallstones slipping into CBD

b) gallstone in cystic duct and getting impacted onto CBD (Mirrizi syndrome)

c) pancreatic calculus obstructing at the ampulla of Vater

2. STRICTURES

a) malignant carcinoma of CBD

b) Benign – surgical trauma

c) primary sclerosing cholangitis

3. TUMOURS OF THE BILIARY TREE

a) periampullary carcinoma

b) carcinoma of head of pancreas

c) cholangiocarcinoma

4. EXTRINSIC PRESSURE ON EXTRAHEPATIC BILIARY TRACT

a) Metastatic lymphnodes near the biliary tract by pressure and later by infiltration

produce obstruction to biliary passages

b) primary lymphnodular disease involving the lymphnodes near biliary pathways –

histiocytic non-Hodgkin's lymphoma

c) Metastatic involvement of the connective tissue of hepatic hilum causing extrinsic

compression on bile ducts

5. MISCELLANEOUS CAUSES

a) parasitic occlusion of CBD – Schistosomiasis

b) Mycotic condition

c) Choledochal cysts

d) Hepatic artery aneurysm

II. INTRAHEPATIC CAUSES

1. INTRAHEPATIC STONE

2. INTRAHEPATIC BILIARY STRICTURES

3. KLATSKIN'S TUMOUR

4. BILIARY DYSPLASIA

a) Congenital hepatic fibrosis

b) Cystic disease of the liver

c) Caroli's disease

5. CONGENITAL AND INFANTILE ATRESIA OF BILE DUCTS

6. ANEURYSM OF BRANCHES OF HEPATIC ARTERY

7. CYSTS OF THE LIVER

a) congenital

b) parasitic

8. PRIMARY AND SECONDARY MALIGNANCIES OF THE LIVER

CLINICAL CLASSIFICATION OF OBSTRUCTIVE BILIARY TRACT DISEASE

Classification proposed by Benjamin (1983) has proved useful in clinical practice. It recognizes four types of biliary obstruction. They are:

TYPE I: COMPLETE

Obstructive – producing progressive jaundice

Eg.:

a) Tumours of head of pancreas

b) Cholangiocarcinoma

c) Ligation of CBD

d) Parenchymal damage to liver

TYPE II: INTERMITTENT

Obstruction which produces symptoms and biochemical changes with or without jaundice

Eg.:

a) choledocholithiasis

b) periampullary carcinoma

c) duodenal diverticula

d) papillomas of bile duct

e) choledochal cysts

f) polycystic liver disease

g) intrabiliary parasite

TYPE III: CHRONIC INCOMPLETE

Obstruction with or without symptoms and biochemical changes eventually producing

pathological changes in bile ducts of liver

Eg.:

a) strictues of CBD

1) congenital

2) traumatic

3) post irradiation

b) stenosed biliary enteric anastamosis

c) stenosis of sphincter of Oddi

d) chronic pancreatitis

e) cystic fibrosis

TYPE IV: SEGMENTAL

Obstruction in which one or more anatomical segments of biliary tree may be obstructed. This

in turn may be complete, intermittent or chronic incomplete.

Eg.:

a) traumatic (including iatrogenic)

b) hepatico choledocholithiasis

c) sclerosing cholangitis

d) cholangio carcinoma

INVESTIGATIONS

BIOCHEMISTRY

Biochemical features of cholestasis are:

1. Conjugated hyperbilirubinemia

2. Elevation of alkaline phosphatase, 5' nucleotidase, gamma glutamyl transpeptidase. Theenzyme 5' nucleotidase is the most reliable since its level is not influenced bybone disease or alcoholism.

3. Minimal or no elevation of serum transaminases

4. Presence of bilirubin in the urine as conjugated bilirubin, which is water soluble andhence filtered by glomeruli

5. Elevation in serum cholesterol and bile acid levels although these are not routinelymeasured inpatients with cholestasis jaundice.

IMAGING TECHNIQUES

PLAIN ABDOMINAL AND CHEST SKIAGRAM

Calcification in the region of gall bladder indicates gall stone. Multiple areas of calcificationin the region of the pancreas are helpful in diagnosing chronic calcific pancreatitis.

ULTRASONOGRAM

This is non-invasive and quick to perform, but requires experience in technique andinterpretation. Extrahepatic biliary obstruction can be diagnosed by demonstration of dilatedbiliary radicals. In experienced hands the accuracy in diagnosing ductal dilatation is over95%. In most iof the cases the cause of biliary obstruction can be traced by ultrasonogram.

Enlargement of head of pancreas is suggestive of carcinoma. Difficulties in achieving adefinite diagnosis arises principally with small lesions at the lower end of common bile ductand which is often obscured by gas in the duodenum or colon. As it does not involveradiation, it can be used safely in pregnancy.

ENDOSCOPIC RETROGRADE CHOLANGIO-PANCREATICOGRAPHY (ERCP)

Upper GI endoscopy with a forward or oblique viewing pan endoscope should be performedin jaundiced patients as significant gastrointestinal pathology is encountered in 25% ofjaundiced patients. This is indicated when obstructing agent is lower down in the CBD. Thisis also ideal when ducts are not dilated or visualization of pancreatic duct or ampulla isrequired. It permits concomitant endoscopic examination and biopsy of lesions encountered during endoscopic examination. Certain lesions can be treated or palliated during thisprocedure like endoscopic stone removal, endoscopic nasobiliary drainage and stent insertionfor inoperable malignant large bile duct obstruction.

ERCP has very low morbidity due topancreatitis (1%) and very low mortality (0.1%).

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAM (PTC)

This is done by injecting the contrast material into the dilated biliary radicles through acannula. This more useful when the obstruction is higher up in the bile or hepatic ducts.

CT findings in pancreatic carcinoma are:

a) Focal mass

b) Pancreatic atrophy/pancreatitis

c) CBD/PD dilatation

d) Vascular enhancement or displacement

e) Regional lymphadenopathy

MAGNETIC RESONANCE CHOLANGIO-PANCREATICOGRAPHY (MRCP)

It is a newer development in MRI. It provides multiplanar, cross-sectional, reconstructiveimage of pancreaticobiliary tree. It is purely diagnostic with no therapeutic interventionpossible. It offers advantage over dynamic CT in suspected hilar cholangiocarcinoma andprimary gall bladder carcinoma. Studies show promises with choledocholithiasis andmalignant bile duct obstruction.

ENDOSCOPIC ULTRASONOGRAPHY

It is in the early stage of development. A useful modality for tumour exclusion whentransabdominal utrasonography or CT has failed and high index of suspicion of carcinomaexists due to elevated tumour markers, prior to elevated tumour markers or ERCP. It canconfirm tumour of 1.2 cm in head of pancreas.

LAPAROSCOPY

Should be routinely used by surgeons in all patients with jaundice. It gives direct visualizationof underlying pathology and is valuable in staging hepatobiliary and pancreatic tumours. Itavoids unnecessary laparotomy for patients with inoperable diseases.

ANGIOGRAPHY

Preoperative angiography is indicated in

1. history of previous major upper abdominal surgeries
2. doubtful resection on clinical and CT appearances
3. when it is anticipated to remove major vascular structures

Angiographic findings in pancreatic carcinoma are:

1. parenchymal hypovascularity
2. angulation of vessels
3. encasement of vessels (arterial/venous)
4. displacement of vessels
5. arterial neovascularity

FINE NEEDLE ASPIRATION CYTOLOGY

A fine needle of 21-23G is passed under guidance. The specimen is expressed on tomicroscopic side, smeared, fixed in 95% alcohol or autospray and stained by modifiedpapanicolaou or other methods. Diagnosis can be made within 20 minutes of obtainingFNAC. Irregularly arranged clusters of cells exhibiting cellular pleomorphism, large vesicularnuclei and prominent nucleoli are seen. However such cytological appearances maysometimes fail to differentiate between adenocarcinoma of gland and lymphoma. Positivediagnosis can be obtained in 87-100% with few false positive results.The earlier the tumour, the smaller it is. It then becomes difficult to obtain diagnosis byneedle biopsy techniques. Complete resection remains the best biopsy. Every surgeon should be prepared to accept occasional benign biopsy report. This technique should be avoided inpotentially respectable tumours with the theoretical possibility of seeding along needle tracks.It is to be noted that it could be on the other hand a very valuable method in elderly frailpatients in whom a surgical palliation is being contemplated. It should also be avoided in ayoung relatively fit patient in whom a histological as opposed to cytological proof ismandatory prior to chemo or radiotherapy'

CARCINOMA OF PANCREAS

It includes carcinoma of the head proper and periampullary region. Almost all carcinoma ofpancreas arise from the ductal epithelium. Only 1% arises from acini. The average age ofpatient is about 60 years, but carcinoma of ampulla the average age is about 5 years less.Males are more affected.

PATHOGENESIS

Incidence of carcinoma of the pancreas has risen steadily over the past 10 years. There aresome factors, which can be considered as initiating or provoking carcinoma of pancreas. Theyare:

1. Cigarette smoking
2. Consumption of coffee
3. Diet rich in fat
4. Chemicals such as beta napthylamine and benzidine
5. Diabetes
6. Carcinogens in duodenal contents refluxing into the pancreatic duct
7. Alcohol consumption

PATHOLOGY

Adenocarcinoma is the predominant lesion often accompanied by extreme fibrous connectivetissue stromal proliferation. The tumours may be mucinous of non-mucin secreting. Only 10%assume an adenosquamous pattern of extreme anaplasia with giant cell formation, numerousmitosis and bizarre pleomorphism. Only 5% arise in cyst and are termed cystadenocarcinoma.

Carcinomas of the ampulla of Vater are columnar cell adenocarcinoma. This neoplasm arisesin duodenal papilla, in the ampulla of Vater or in the duodenal mucosa adjacent to the papillathere may be an area of pancreatitis in the head of pancreas. The primary lesion is so smallthat it is difficult to palpate. In such carcinoma, jaundice may not be progressive as recurrentsloughing of the central portion of the tumour will relieve obstruction of bile duct and jaundice becomes intermittent.

CLINICAL FEATURES

Carcinoma of the head of the pancreas usually presents with painless progressive obstructivejaundice. Progressive jaundice is usually associated with pruritis due to the presence of bilesalts in blood. The jaundice usually progresses steadily until the patient is almost green incolour. In case of periampullary carcinoma, the jaundice may be intermittent. Pain is not a marked feature. Patient may complain of dull and aching pain in the epigastrium. Pain is oftenrelieved by sitting in hunched position and is aggravated by supine position. Eating mayaggravate pain. Weight loss is the single most common symptom of carcinoma of thepancreas irrespective of the position of the tumour. Diarrhea with pale and foul smelling stool is sometimes a feature of periampullary carcinoma. There may be steatorrhoea due to enzymedeficiency.On examination, jaundice is the main sign. A palpable distended gall bladder is detected in60% of cases. Enlargement of liver is found in slightly more than half the cases. In carcinomaof head of pancreas it is often

due to biliary obstruction.Carcinoma of ampulla of Vater shows a few peculiar symptoms and signs. Pain is lessfrequent in this condition but when present is apt to be more colicky in nature. Jaundice isintermittent. Chills and fever are not uncommon due to associated cholangitis. Hematemesisand melena occasionally occurs in late cases as a result of direct invasion of duodenal or
gastric mucosa by tumour and portal hypertension secondary to splenic or portal veincompression by the tumour.

CHOLANGIOCARCINOMA

The reported autopsy incidence of malignant bile duct tumour ranges from 0.01-0.5%. Thereis slight preponderance of male (1.5:1). The age at presentation varies but the peak incidenceis in sixth decade. The etiology of bile duct cancer is unknown. Cholangiocarcinoma is seenwith increasing frequency in parasitic infestation of biliary tree, cystic disease of biliary tract,
chronic typhoid carriers, and ulcerative colitis and sclerosing cholangitis.

PATHOLOGY

Tumours are best classified into the anatomical site of origin

1. Intrahepatic tumour from minor hepatic ducts

2. Proximal from right or left hepatic ducts, cystic duct and it confluence with CBD.

3. Middle from the distal common hepatic duct, cystic and its confluence with CBD.

4. Distal from the distal common bile duct and periampullary region.

Tumours of the minor hepatic ducts are often diffuse (multicentric) and difficult todifferentiate from primary hepatocellular carcinoma. The gross appearance of cholangiocarcinoma assumes one of the three forms. They are:

1. Strictures

2. Nodular

3. Papillary

The majority of tumours are adenocarcinoma of varying origin. All cholangiocarcinomas havea special predilection for perineural spread and do not metastasise beyond the liver. The bestprognosis is encountered after resection especially of distal and periampullary lesions.

CLINICAL FEATURES

The main presentation (90%) is with obstructive jaundice which is progressive andaccompanied by itching and anorexia. Dull upper abdominal pain is a frequent symptom.

Some patients present acutely with cholangitis. Physical examination reveals hepatomegaly.Anemia is present in patients with papillary tumours especially at the lower end of bile ductand periampullary region. It is caused by chronic blood loss. The feces of these patients havea characteristic silvery appearance due to combination of steatorrhoea and altered blood. A palpable gall bladder is present in patients with distal tumours.

PRE-OPERATIVE PREPARATION

1. All jaundiced patients must be kept in a good state of nutrition and hydration withsupplemental intravenous fluids, elemental diet and multivitamins as deemednecessary. Renal failure due to hypovolemia is a tremendous hazard post-operativelyand a continuous diuresis is maintained at all times. If the patient is grosslymalnourished, a period of parenteral hyperalimentation both before and after operationmay be of additional benefit.

2. Blood clotting deficiencies must be corrected. Anaemia is corrected by blood transfusions. Daily injection of Vit K is administered, preferably 4-5 days prior tooperation. Six units of fresh frozen plasma, six units of platelets and atleast six units ofblood should be made available in operating room.

3. Cardiopulmonary function should be assessed by pulmonary function tests, chest Xray and ECG. smoking is prohibited. Intensive pulmonary physiotherapy, activemobilization and leg exercises are strongly encouraged post operatively.

4. Antibiotic prophylaxis should be given since there is impaired wound healing due todepressed immune function.

5. Nutritional status to be assessed and supported as there is impaired wound healing dueto decreased fibroblastic activity and general protein and calorie malnutrition.

6. If patient is critically ill with one or more of the following parameters,

a) highly elevated serum bilirubin (>12 mg%)

b) sepsis

c) hepatorenal failure

d) severe cardiopulmonary disease

e) malnutrition

a percutaneous transhepatic biliary drainage or endoscopic decompression should beattempted to tide over the patient for 2-3 weeks before major surgery. If the techniqueof percutaneous biliary drainage or endoscopic stenting is not available, a simplecholecystectomy or T-tube drainage of CBD may be undertaken.

TREATMENT OF MALIGNANT OBSTRUCTIVE JAUNDICE

Treatment can be either

1. Curative

2. Palliative

CURATIVE TREATMENT

Surgery is now considered as the gold standard for treatment of malignant obstructivejaundice against which all other new modalities are considered. Halsted performed firstcurative and successful resection of periampullary carcinoma at John Hopkins Hospital in1898. He performed local resection of ampullary tumour. Presently standard resection for periampullary carcinoma and head of pancreas tumours involves a pancreaticoduodenectomy,first performed successfully by Kausch in 1909 and

popularized by Whipple 1935. The gallbladder, CBD, entire duodenum, head of pancreas, pancreas upto the level of superiormesenteric vein, pylorus and distal stomach are resected. Restoration of gastrointestinal
continuity utilizes the proximal jejunum, brought out through the transverse mesocolon forpancreaticojejunostomy, hepaticojejunostomy and gastrojejunostomy. The standard Whippleresection remains the classic thrrapy for these tumours and can be successfully performed inexperienced hand swith mortality less than 5%. A modification of standard Whipple resection,
the pylorus preserving pancreaticoduodenectomy has gained popularity in recent years. Thismodification eliminates gastric resection and leaves a 2cm cuff of duodenum for entericreconstruction as duodenojejunostomy.

PALLIATIVE SURGERY

Palliative surgery for periampullary carcinoma is performed in patients with unresectabledisease discovered at the time of laparatomy or in patients with prohibitive risk for resectionaltherapy (advanced age, limited cardiopulmonary reserve and also poorly alleviated nonoperatively).

1. Relief of jaundice, pruritis and impending cholangitis: Biliary tract decompression canbe done either by cholecystojejunostomy or by hepaticojejunostomy (each with
diverting enterostomy) depending on whether the cystic duct is widely patent and is infull communication with the biliary tree proximal to the obstructing cancer.

2. Relief of duodenal obstruction: If the patient lives for more than few months, duodenalobstruction usually occurs. It is therefore advisable to perform a gastrojejunostomy atthe primary operation.

NON OPERATIVE MANAGEMENT

When a patient is unfit or refuses surgery an alternative method of palliation of the jaundice isby endoscopic sphincterotomy and placement of biliary stent. This approach does not relieveany additional obstruction, which may be present. If patient survives for more than a fewmonths, recurrent cholangitis associated with stent blockage is a problem that necessitates

regular endoscopic removal and replacement of the stent.

Percutaneous transhepatic placement of internal expandable metal stent is being tried byinteventional radiologist and offers yet another option for palliation of the jaundiced patientwith malignant biliary tract obstruction.

TREATMENT OF CHOLANGIOCARCINOMA

Resection is the best method of treatment and is indicated for all operative tumours in fitindividuals. The reported respectable rate varies but averages 20%. The benefits of resectionare:

1. The possibility of cure or long term survival especially for distal bile duct tumours

2. Resection provides the best form of palliation in terms of duration and freedom frominfective complications.The surgical procedure depends on the location of tumour. For hilar lesions, an anterior

segmentectomy IV provides good access to confluence, allowing good clearance proximal tothe tumour and facilitates hepaticojejunostomy. When the tumour extends along the right orleft duct with extension to respective lobe, the resection includes a lobectomy in continuation
with main tumour mass. Middle tumours are excised from just below the confluence down tothe duodenum together with associated pericholedochal lymphnodes. The surgical treatmentof periampullary tumours is pancreaticoduodenectomy. The results of hepatic transplantation
for cholangiocarcinoma (diffuse intrahepatic type) have been disappointing.

PALLIATIVE SURGERY

If tumour is inoperable, a bilio-enteric bypass is performed. Anastamosis of Roux loop tosegment III duct using the round ligament approach gives the best results for inoperable hilarlesions. Longmire operation in which anastamosis of the segment III duct to Roux loop ofjejunum after left lateral segmentectomy and Smith operation used to be done earlier, butthere is no added advantage to these procedures. A cholecystojejunostomy is performed forinoperable distal tumours. A gastrojejunostomy is added if duodenal obstruction is present or considered imminent in patient with periampullary tumour.

NON OPERATIVE MANAGEMENT

In patients who are considered inoperable as preoperative assessment and those who are too

old and frail, palliation of jaundice is achieved by percutaneous transhepatic or endoscopicstenting. The endoprosthesis has to be large 8-10 FG and may require replacement if itbecomes blocked. Recently self-expandable stainless steel wire endoprosthesis have beenintroduced in management of patients with malignant biliary strictures. Other causes ofmalignant obstructive jaundice are due to extrinsic compression on the biliary tract bytumours, Metastatic lymphnodes near the biliary tract by pressure and later by infiltration andprimary lymphonodular disease involving lymph nodes near biliary pathways. Treatment isprimarily to relieve obstructive jaundice and troublesome pruritis and steatorrhoea

Choledocholithiasis

1. Endoscopic sphincterotomy, stone extraction/CBD stenting

followed by Lap/Open cholecystectomy

2. Lap/Open Cholecystectomy followed by Lap/Open CBD

Exploration

CBD Exploration

First surgical exploration of the CBD was done in 1980 byLudwig Courvoisier.

Indications

1. PREV.HISTORY OF JAUNDICE / CHOLANGITIS /

PANCREATITIS

2. PALPABLE STONES IN CBD

3.DILATED CBD

4. MULTIPLE SMALL STONE

Lap CBD exploration most commonly done

Either trancystic or transductal

Transductal

• Stones >6mm

• Intrahepatic stones

• Cystic duct diameter<4mm

• Cystic duct entrance either posterioror distal

T-Tube

• For decompression if CBD not cleared

• Later study of biliary system

• Access to biliary system for recurrent stones

Placing 'T' Tube:

• Shorten limbs & remove part of wall

• Allows sphincter edema to settle

• 14 F size

• Tract for future intervention if retained stones are

detected

• T tube cholangiogram 7 – 8 days

• If normal – remove > 12 days

• Retained stone – keep ‘T’ tube

• Intervention 5 – 6 wks. Later

Retained / Recurrent Stones:

Retained - detected in a short time after surgery

Recurrent - diagnosed months or years later

Choice of treatment

-Clinical presentation

-Presence of T tube

- Endoscopic expertise

T tube in situ:

-OBSERVATION

-MECHANICAL EXTRACTION- BURHENNE

TECHNIQUE

-DISSOLUTION-Mono Octanoin instillation

-CHOLEDOCHOSCOPIC CLEARANCE

-LITHOTRIPSY

Endoscopic treatment:

• ERCP/Endoscopic sphincterotomy

85 – 95% success

Difficult

• Stones >2cms

• Distal stricture

• After Billroth II anastomosis

If non operative treatment fails

OPEN OR LAPARASCOPIC CBD EXPLORATION

3. Biliary drainage procedures:

Surgical biliary drainage procedures must be considered in the following situations

• Multiple stones

• Incomplete removal of all stones

• Impacted, irremovable distal bile duct stones

• Markedly dilated common bile duct

• Distal bile duct obstruction from tumour or stricture

• Reoccurrence after previous bile duct exploration

Methods of surgical drainage include

• Trans duodenal sphincteroplasty

• Choledochoduodenostomy

• Choledochojejunostomy

Choledochol cyst

Type-1: solitary fusiform extra-hepatic cyst:

Excision + roux-en-y hepaticojejunostomy

Type 2:- Diverticular dilation of extra hepatic biliary tree

Excision of dilated diverticulum

Closure over T-tube

Type3:-Cystic dilation of intraduodenal portion of CBD

(choledochocele)

Choledochocele <3cms- Endoscopic sphincterotomy

Choledochocele >3cms- Trans duodenal excision

Type 4:-

4A: Multiple cysts both in extra & intrahepatic biliary tree

Extra hepatic: excision with hepatico jejunostomy

Intra hepatic: hepatic resection

4B: Multiple extra hepatic cysts

Excision with hepatico jejunostomy

Type 5:-

Intrahepatic multiple cysts associated with cirrhosis or

periportal fibrosis

Confined to single lobe: Hepatic lobectomyMultilobar associated with hepatic failure, cirrhosis, andportal hypertension: liver transplantation

4.AIMS OF THE STUDY

AIMS & OBJECTIVES:

1. To analyse the incidence of benign and malignant causes for obstructive jaundice in our hospital.

2. To analyse the age and sex distribution.

3. To study various clinical presentations.

4. To evaluate various management modalities.

5. To evaluate the histopathology of resected specimen.

5.MATERIALS AND METHODS:

The study is to be carried out in Govt .Stanley Medical college Hospital, Chennai .

This is a facility based prospective descriptive study involving all patients with obstructive jaundice.

The relevant data shall be collected by using:

- Detailed history
- Hematological investigations: complete hemogram , liver function tests including serum alkaline phosphatase serum proteins and albumin, blood urea, serum electrolytes.
- Radiological investigations like as USG Abdomen and CECT abdomen scan to find malignancy when required
- MRCP and ERCP to asses pathology of biliary tree .
- Histopathological examination for the patients who underwent surgery
- Follow up of non surgical method s as stenting etc
- All the recorded variables will be tabulated and analysed with multivariate analysis and chi square test

SOFTWARE USED ; SPSS ver20.0

SETTING : Govt.Stanley Medical College

Chennai-1.

DESIGN OF STUDY : FACILITY BASED PROSPECTIVE

DESCRIPTIVE STUDY

PERIOD OF STUDY : NOVEMBER 2013 TO DECEMBER 2015]

INCLUSION CRITERIA:

1. All patients with obstructive jaundice due to extra hepatic biliary obstruction as diagnosed by MRCP

and ERCP

EXCLUSION CRITERIA:

1. Patients with obstructive jaundice. due to intra hepatic calculi and stricture 2. Patients with hemolytic and hepatocellular jaundice. 3.Patient aged <20 yrs and >80 yrs of age

SAMPLE SIZE : 50

6.TABLES AND CHARTS

TABLE .1.AGE DISTRIBUTION

Age group (in years)	No of patients	Percentage of patients
21-30	3	6%
31-40	4	8%
41-50	10	20%
51-60	19	38%
61-70	11	11%
71-80	3	6%

Most common in 5^{th} and 6^{th} decade of life

AGE DISTRIBUTION

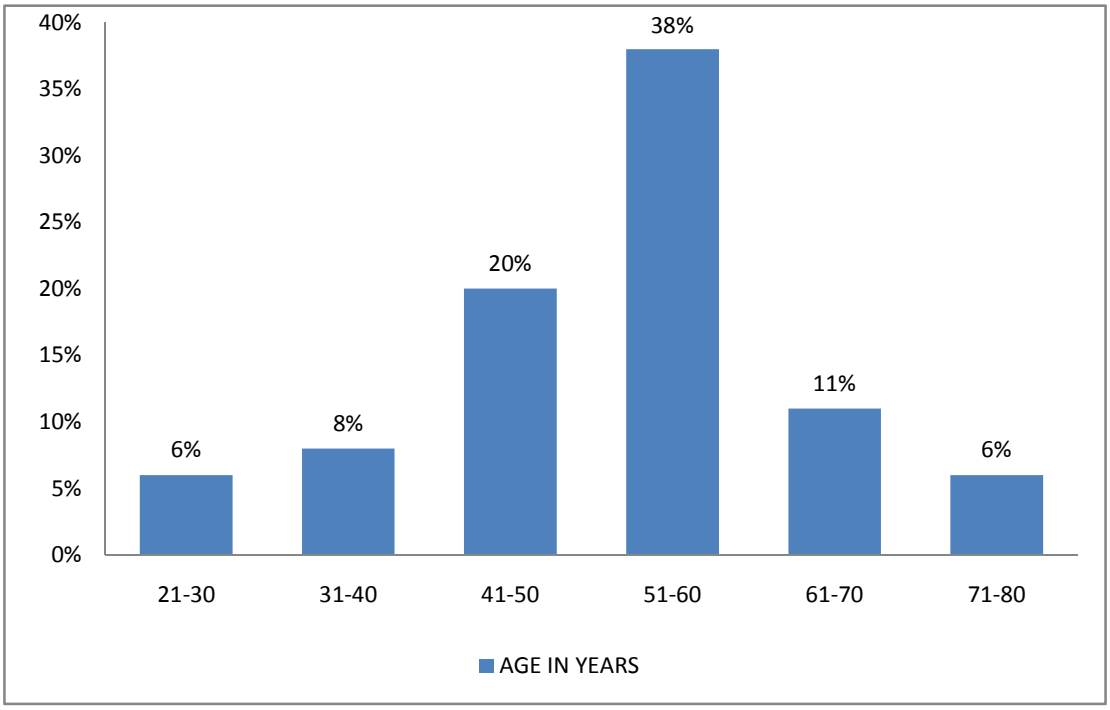


TABLE .2

SEX DISTRIBUTION

SEX	**NUMBER**	**PERCENTAGE**
MALE	**19**	**38%**
FEMALE	31	62%

In our study FEMALES are affected more than MALES about

Male :Female Ratio→2:3

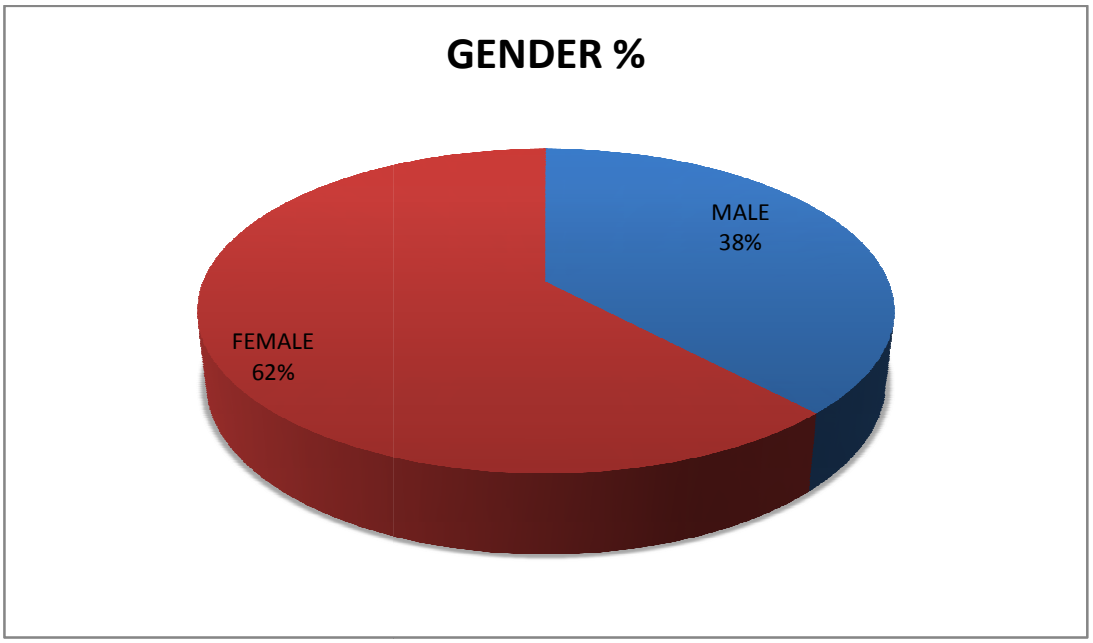
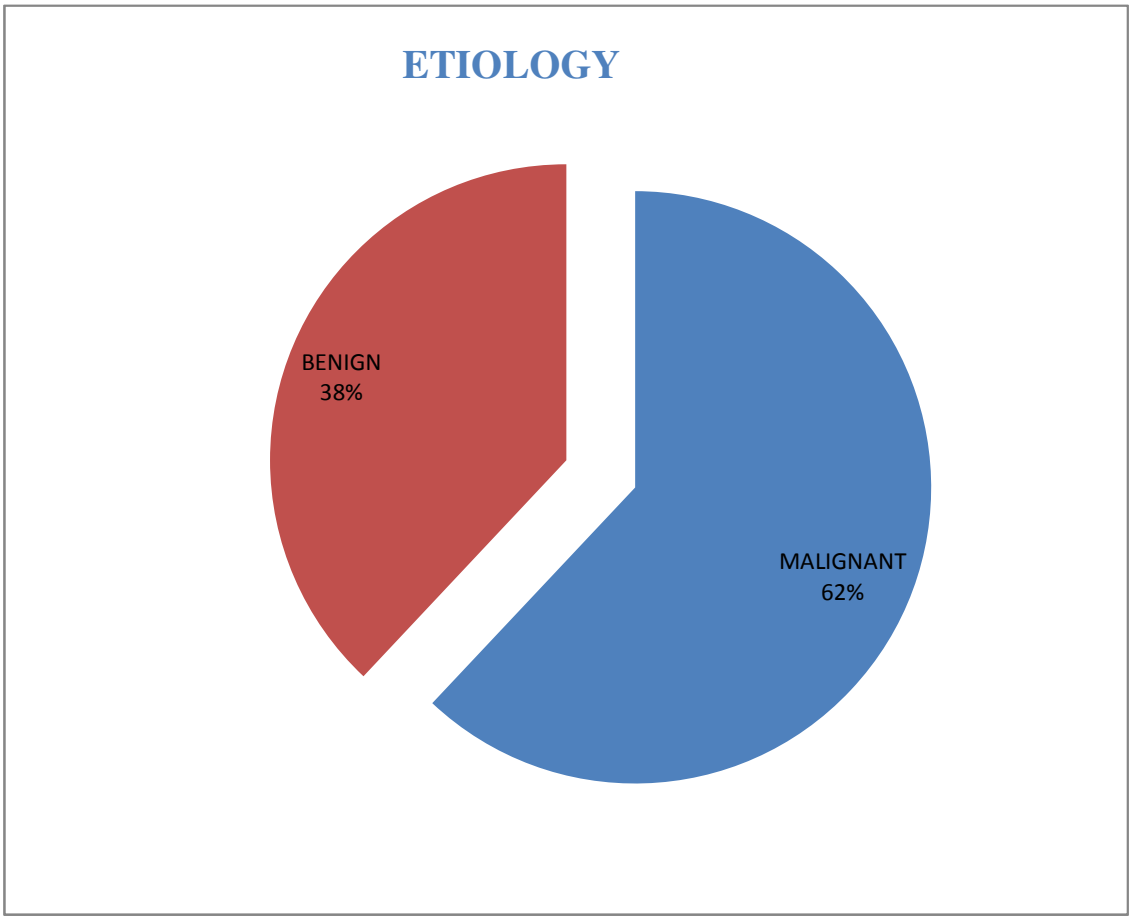


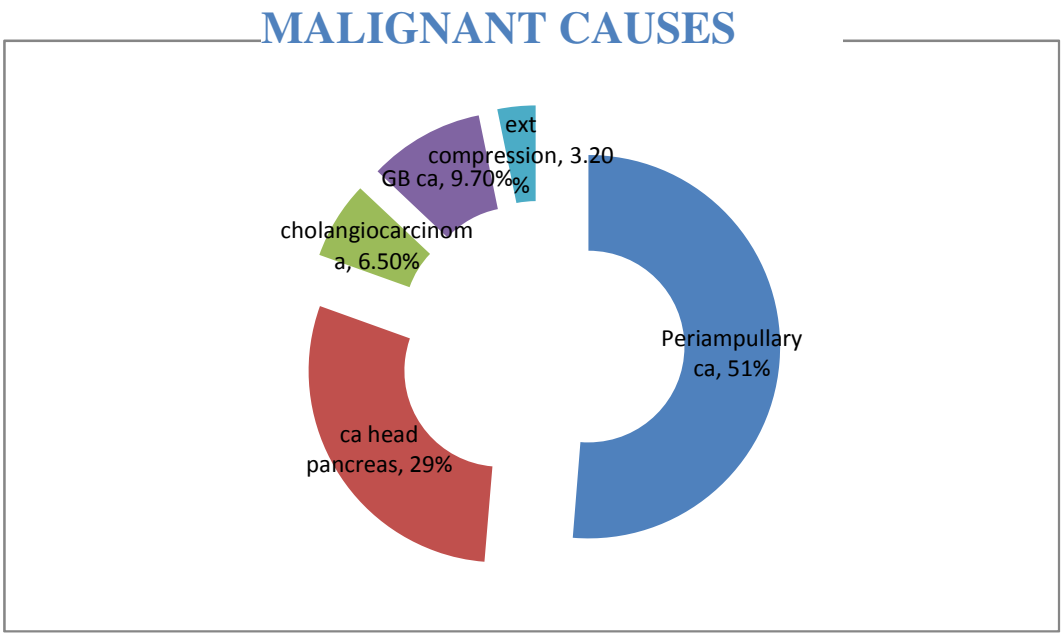
TABLE 3

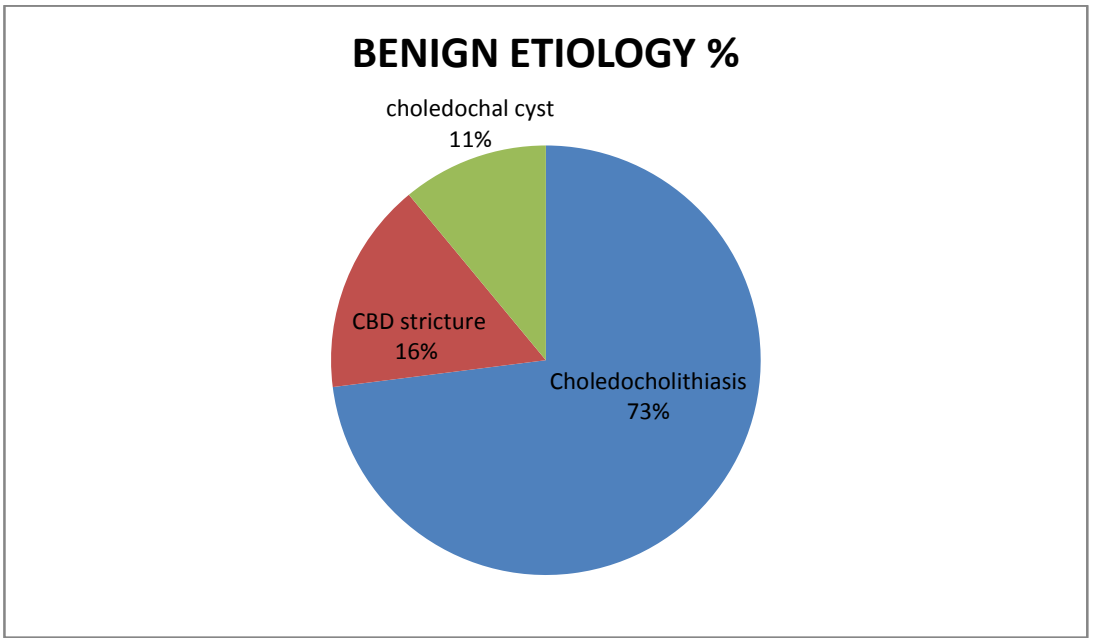
ETIOLOGICAL CLASSIFICATION

S. No	ETIOLOGY OF OBSTRUCTIVE JAUNDICE	NO OF PATIENTS	% OF PATIENTS
	MALIGNANCY	**31**	**62%**
1.	Periampullary carcinoma	16	32%
2.	Carcinoma head of pancreas	9	18%
3.	cholangiocarcinoma	2	4%
4.	Carcinoma gall bladder	3	6%
5.	Extra neous compression	1	2%
	BENIGN	**19**	**38%**
1.	Choledocholithiasis	14	28%
2.	CBD Stricture	3	6%
3.	Choledochal cyst	2	4%

Most common cause for obstructive jaundice is MALIGNANCY >BENIGN Condition forming around 68% of cases especially Carcinoma Periampullary region forms 32% of patient group


ETIOLOGY
BENIGN
38%
MALIGNANT
62%

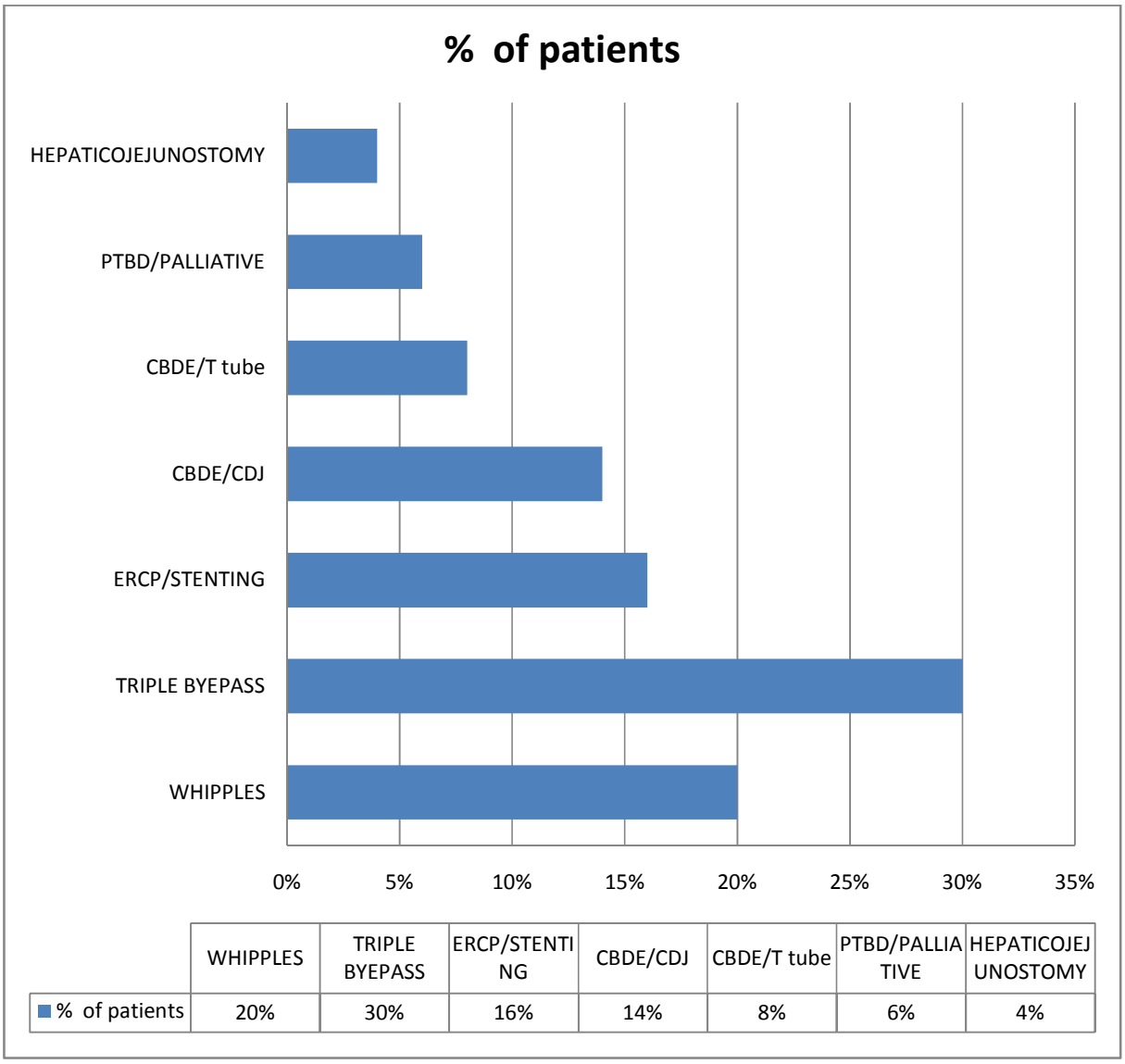

MALIGNANT CAUSES
ext
compression, 3.20
%
GB ca, 9.70%
cholangiocarcinom
a, 6.50%
Periampullary
ca, 51%
ca head
pancreas, 29%



In case of benign etiology choledocholithiasis form the main role of around 73%.

TABLE 3
MANAGEMENT MODALITIES

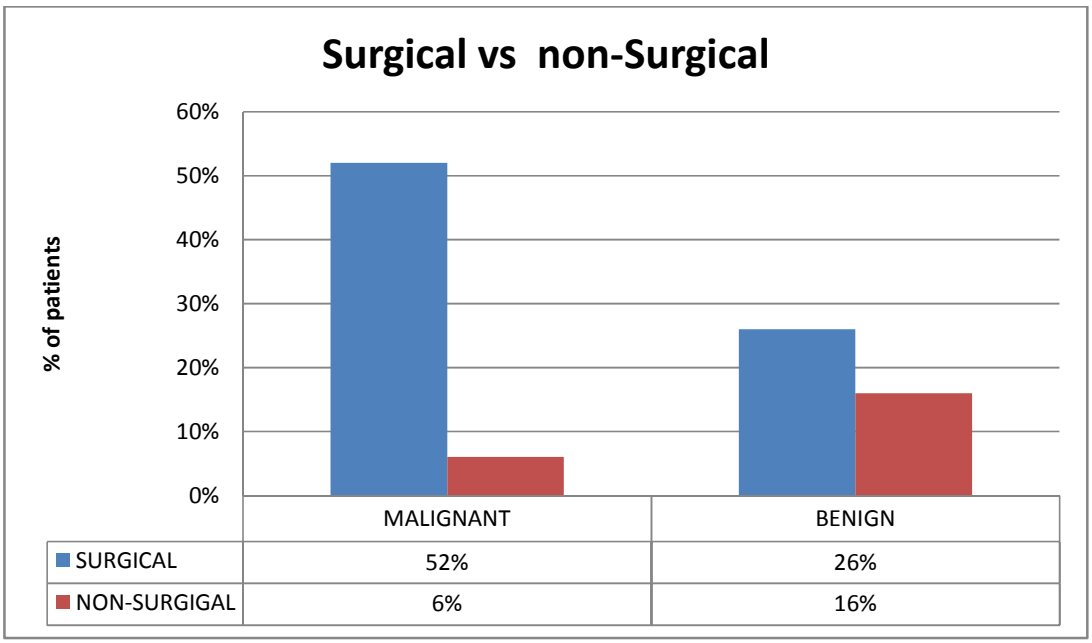
S NO	**MANAGEMENT**	NO OF PATIENTS	% OF PATIENTS
1.	TRIPLE BYEPASS	15	30%
2.	WHIPPLES PROCEDURE	10	20%
3.	ERCP/STENTING	8	16%
4.	CBDE+CDJ	7	14%
5.	CBDE+T tube	4	8%
6.	PTBD+PALLIATIVE	3	6%
7.	HEPATICOJEJUNOSTOMY	2	4%



	WHIPPLES	TRIPLE BYEPASS	ERCP/STENTING	CBDE/CDJ	CBDE/T tube	PTBD/PALLIATIVE	HEPATICOJEJUNOSTOMY
% of patients	20%	30%	16%	14%	8%	6%	4%

In case of management ,surgery forms major role in both benign as well as malignant cases.Around 78% of cases are treated by surgery and remaining 22% cases are treated by non surgical modalities

MANAGEMENT STRATERGY



7.RESULTS AND OBSERVATIONS

We studied 50 patients of Obstructive jaundice with extra hepatic cause in our wards in Govt.stanley medical college chennai 01, from november 2013 to June 2015.

Among 50 patients 32% were male and 68% were female. The mean age group was 54.19 yrs. The age range from 21 to 84 years and more common in fifth and sixth decade of life.

The average duration of illness was 4.8 months, the range being 10 days to 12 months. The mean duration of hospital stay was 18days that range between 15days to 60 days.

All patients had icterus (100%). 55% of patients had pain abdomen, of which 42% of patients had typical colicky type of abdominal pain. 44% of patients had fever, of which 31% of patients were associated with chills and rigors.

Symptoms of complete biliary tract obstruction, clay coloured stools and high coloured urine presented in 30% of patients. Cachexia was seen in 29% of patients.

Gall Bladder was palpable in 52% of patients, of which most were due to pancreatic and Periampullary malignancies.

The mean serum bilirubin value was 12.5 mg%. The range between 2.0-28 mg%. The average ALP value was 420.85 IU, and the range between 108-1230 IU. Urine examinations showed absent in

urobilinogen in 42 % of patients. Serum albumin range was 2.5-5.5 gm. %. In More than 50 % of patients, the A: G ratio was reversed.

Ultra sonogram revealed IHBR dilatation in 90% of pts.

Therapeutic ERCP done in patients of CBD stone disease. After therapeutic ERCP laparoscopic/open cholecystectomy was done in all 16%patients.

Preoperatively all patients received three doses of Vit K and fresh frozen plasma in selective patients. Coagulation profile was monitored by measuring PT and INR.

In our study we observed the most common cause of biliary tract obstruction was due to periampullary carcinoma accounting for.Among these most of the patients were females (16pts., 32%). The second most common causewas choledocholithiasis(28%), followed by ca head of pancreas (18%). More common in fifth decade oflife.

Other rare causes of obstructive jaundice observed in our study were Stricture CBD (6%), Cholangiocarcinoma (4%), Carcinoma Gall Bladder (6%), Choledochol cyst (4%) and carcinoma Stomach with porta hepatis metastasis (2%).

CBD stones are treated by therapeutic ERCP/Stenting, CBD Exploration and biliary enteric anastomosis or T Tube Drainage.

Among these CBDE/T tube/ CDJ was most commonly done (20%).

Among the malignant causes, curative resection (Whipples

procedure) was done in 4 patients of Ca Head of Pancreas and 6 patients of Periampullary carcinoma (20%). Most of the patients with Ca head of Pancreas and periampullary carcinoma were locally advanced and treated byPalliative bypass procedure (30%).

2 patients expired in our study group. All expired patients had biliary tract obstruction due to malignant aetiology. The most common complication noticed in operated patients was biliary fistula. Fistula is more common following palliative procedure for malignant aetiology. Patients with benign diseases are on regular follow up and they doing well.

The histopathology report of pancreatic cancer consists of well differentiated adenocarcinoma (30%), moderately differentiated in (30%) and poorly differentiated (40%).

None of the patients withcarcinoma Gall bladder were operable. Biliary obstructions in thesepatients were relieved by ERCPStenting/palliative chemotherapy.

In our study 3 of patients were due to stricture in the biliary tract. Of these 2 patient had terminal CBD stricture underwent non surgical procedure as STENTING and 1 patient had mid CBD stricture underwent choledochojejunostomy . For CBD stone, for which CBD explorationandCholedochojejunostomy was done. In our study all the patients withstricture was due to chronic calcific pancreatitis/ endoscopic procedure.

Out of 50 patients, 4% patients were due to Choledochol cyst. For which Total cyst excision and biliary enteric anastomosis was done.

8.DISCUSSION

Jaundice is a most challenging problem for any person, more so when people are ignorant of the on-going severe underlying disease.Because of the self-medication and the natural treatment the presentation is very late in suffered patients. Specific symptoms will not occur in early stage of the disease. It will occur after the disease becomes locally advanced or involving adjacent vital structures.

Comparing the other studies done elsewhere, the observation in our study implies, the overall incidence of obstructive jaundice was same in both male and female. The mean age of incidence of surgical jaundice is 51 yrs.But the incidence of Periampullary carcinoma and choledocholithiasis was more common infemales. The most common cause of malignant obstructive jaundicewas periampullary carcinoma, which is more common in femalepopulation especially in fifth and sixth decade of life.

The second most common cause of malignant obstructive jaundice was carcinoma head of pancreas, which was more common in female population.

The lowest age noted for a female patient is 21yrs,were diagnosed to be Choledochol cyst.underwent Excision and choledochojejunostomy was done

Comparing with S.Agal et al of Mumbai who studied 62 cases of malignant aetiology and M.Kannan et al of Chennai who

studied 455 cases of both benign and malignant etiology there is more or less equal age incidence.

The gallbladder felt in 52% of our patients while in Benjamin series it was palpable in 50% of the icteric patients and 62.20% of those with pancreatic malignancies.

Evaluation of obstructive jaundice is common but challenging radiological problem. The aim of the imaging is to diagnose biliary obstruction by identifying dilatation of intra and extra-hepatic biliary channels; to delineate the level of obstruction.

Ultrasonography is widely available, non-invasive and radiation free imaging modality. It is the initial modality for the detection of obstruction in the biliary tree.

Ultrasound was performed in all our patients. It showed dilatation of intrahepatic biliary radicles in 84% of patients.

CBD stones are treated by therapeutic ERCP/Stenting, CBD Exploration and biliary enteric anastomosis or T Tube Drainage. Among these CDJ was most commonly done (12%)

Comparing to other studies of Benjamin and Popper, our study revealed same curative rates in the management of other benign extrahepatic biliary tract obstructive lesions such as stricture of the Common Bile Duct and Choledochal cyst.

In our study, we did not perform any method of preoperative

biliary drainage for any amount of bilirubin levels mainly in patients with malignant cause of biliary obstruction since various studies have shown no difference in the survival benefits with this procedure

We had 5 deaths in the follow up and those under evaluation. These patients were mainly in their advanced stage of their disease and the underlying pathology was mostly advanced carcinoma CBD, carcinoma of the gallbladder, Pancreatic and peri ampullary malignancies.

9.CONCLUSION

- Mostcommon etiology for obstructive jaundice is due to malignant pathology than benign disease.
- The maximum of age incidence is between 51and 60 years (38%)
- Median age is 51.9 yrs
- Male : Female ratio is 2:3
- There is a significant increase in the incidence of malignant obstructive jaundice
- The most common cause of obstructive jaundice isPeriampullary carcinoma followed by Choledocholithiasis
- Periampullary carcinoma was most common in females & most of them in the late fifth and sixth decade oflife.
- Choledocholithiasis was also more common in females.
- Carcinoma head of Pancreas was more common in femalepopulation .
- Most of the malignant cases Presented in late stages and underwent bypass procedures more than resection
- Among the malignant causes, curative resection (Whipplesprocedure) was done in 4 patients of Ca Head of Pancreas and

  6 patients of Periampullary carcinoma (20%).
- Most of the patientswith Ca head of Pancreas and periampullary carcinoma were locally advanced and treated by Palliative bypass procedure (30%).

- A palliative Cholecystojejunostomy with gastrojejunostomy tops the list of operativeprocedures

- Chronic calcific pancreatitis forms as predisposing factor for developing carcinoma head of pancreas

- Biliary tract obstruction due to metastasis is not uncommon.
- Palpable Gall bladder (52%) indicates the etiology to be malignant
- USG followed by MRCP/ERCP and CECT scan are the
    - investigation of choice
- Patients with benign pathology had a better outcome and cure Rate
- Patients with carcinoma gall bladder were mostly inoperable, and
    - underwent palliative treatment only
- The preoperative biliary drainage does not have any survivalBenefit.
- 100% of patients complained jaundice, weight loss and anorexia.
- Mortality due to palliative procedures was 7% and morbidity patterns of wound infection is 10%,delayed gastric emptying is 6%.
- Median hospital stay for palliative procedures was 16 days.
    - Mortality rate following Whipple's procedure was 7.8%

ABBREVIATIONS

GB-Gall bladder

CBD - Common Bile Duct

IHBR - Intra Hepatic Biliary Radicals

ERCP - Endoscopic Retrograde Cholangio Pancreatography

MRCP - Magnetic Resonance Cholangio Pancreatography

PTC - Percutaneous Transhepatic Cholangiography

PTBD-Percutanaeous transhepatic biliary drainage

CBDE - Common Bile Duct Exploration

CCJ/CDJ –Cholecystojejunostomy/Choledochojejunostomy

HJ - Hepaticojejunostomy

11. PROFORMA

Name: IP Number:

Age: Sex:

Address: Unit:

Socio Economic Status:

Date of Admission: Date of Discharge:

Duration of illness;

Symptoms:

Jaundice

Abdominal pain

Fever

Clay coloured stools

High coloured urine

Anorexia

Malena

Steatorrhoea

Pruritus

Loss of weight & appetite

Past History:

Chronic calcific pancreatitis, Diabetes mellitus,

previous

surgery, Blood Transfusion, Previous Drug intake

Personal History:

Dietary habit, Alcoholism, Smoking and Exposure to chemical carcinogen.

Family History:

h/o Jaundice and malignancy

General Examination:

Built: Pallor: Hydration:

Icterus: Scratch marks: Pedal oedema:

BP: Pulse rate:

Signs:

Palpable Gall Bladder

Hepatomegaly

Tenderness

Ascites

Abdominal mass

Signs of Liver cell failure:

Investigations:

Liver function tests

Renal function tests

Complete haemogram

Bleeding time, Clotting time, PT, INR

Urine bile salt, bile pigment, urobilinogen

USG, CECT abdomen scan and MRCP,ERCP

Preoperative preparation:

Vit K, IV Fluids, Antibiotics, Fresh Frozen Plasma.

Preoperative Decompression Procedures:

Surgical Procedure:

Curative:

Palliative:

Per-Op Findings:

Metastasis:

Regional lymph nodes, liver, peritoneum and other sites

Post-Operative recovery & Complications:

Histopathological Report.

CONSENT FORM

STUDY TITLE :1. **Clinical study on etiology and management of obstructive jaundice due to extrahepatic biliary obstruction**

STUDY CENTRE :Govt.stanley medical college, Chennai-1

PARTICIPANT NAME : **AGE:** **SEX**: **J.D.NO**

I confirm that I have understood the purpose of procedure for the above study, I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I understand that the investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties of published, unless as required under the law. I agree not to restrict the use of any results that arise from the study.

I hereby consent to participate in this study of

'Clinical study on etiology and management of obstructive jaundice due to extrahepatic biliary obstruction'

Signature of Investigator: Place :

Date :

Study Investigators Name Institution

Signature / Thumb Impression of patient

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Clinical study on Etiology and management of obstructive Jaundice due to Extrahepatic biliary obstruction .

Principal Investigator : Dr. Saravana Kumar.A

Designation : PG M S (General Surgery)

Department : Department of General Surgery
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.02.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

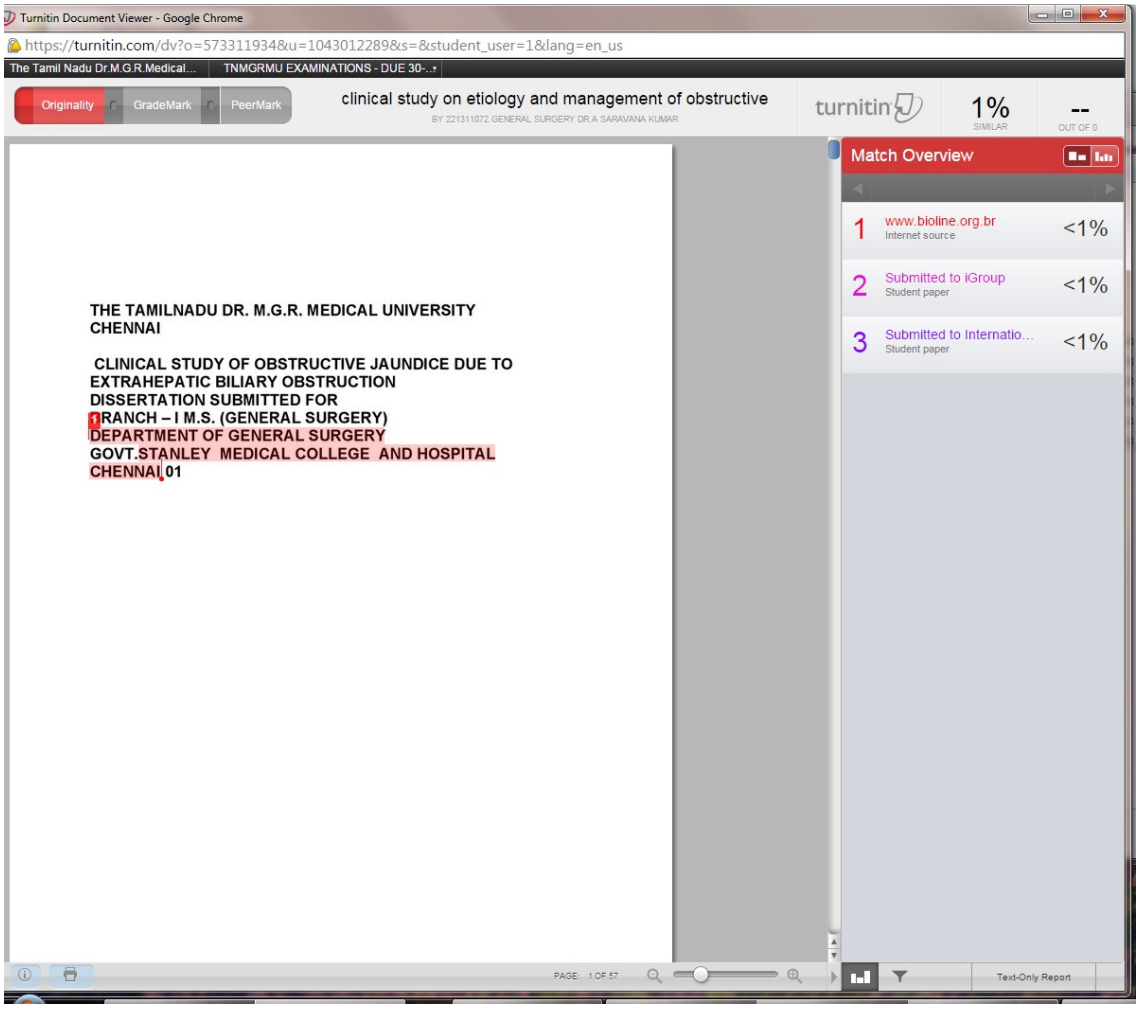
The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.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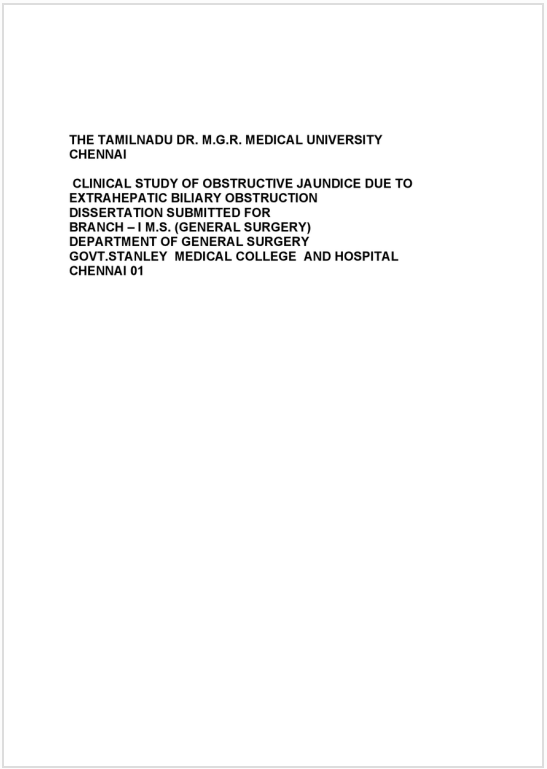

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